Thesis.

R H E U M A T I S M.

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R H E U M A T I S M.

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D E F I N I T I O N.

In remote ages the term "rheumatism",- derived from the Greek "reo, I flow,- was applied to an alleged acrid humor generated in the brain or some other part of the body; from the latter it was supposed to flow in a catarrhal flux,which produced internal or articular inflammations when pent up. The name,however, is now used to somewhat vaguely and unscientifically a variety of conditions,which nonetheless have three things in common,namely,general or constitutional symptoms,largely febrile or toxic;localisation of inflammatory lesions in the joints and sometimes in the muscles or skin;and a tendency to certain visceral inflammatory complications,notably in the heart and serious membranes, and sometimes the tonsils.Thepopular use of the term rheumatism or its derivatives in conjunction with other diseases,such as rheumatic gout,rheumatoid arthritis, and gonorrhoeal rheumatism,is misleading by implication of an etiological relationship which does not exist. Whether they have a common antecedent diathesis or not, there is little or nothing in common in the pathological anatomy or symptoms of gout and rheumatism,or of arthritis deformans and rheumatism,and the so-called gonorrhoeal rheumatism is not a rheumatism at all,but a form of specific septicaemia with localised articular manifestations.

N O M E N C L A T U R E.

The derivation of the word rheumatism has already been given;and we may here further note the fact that it has been loosely applied to numerous conditions which are not rheumatic at all.Some of the qualifying terms are as follows:- Apoplectic rheumatism is rheumatism complicated with apoplexy,due to congestion of the brain. Articular rheumatism is the common form of the disease. Cerebral rheumatism is the disease in association with well-marked cerebral symptoms.Blenorrhagic rheumatism is synonymous with gonorrhoeal rheumatism. Diaphragmatic rheumatism is a self-interpreting term. Encephalic rheumatism is an optional designation of cerebral rheumatism.Epidemic muscular rheumatism is the muscular variety occurring in epidemic form.Gonorrhoeal rheumatism is that variety of the disease due to the machinations of the gonococcus.Gouty rheumatism is synonymous with rheumatoid arthritis. Rheumatism of

the gullet is a functional condition of the oesophagus causing painful deglutition.Rheumatism of the heart is a cardiac affection occurring in the course of rheumatism and synonymous with angina pectoris.Heberden's rheumatism is the articular disease exhibiting a tendency to haemorrhage.Infectious rheumatism is the articular manifestation of a general infection.Muscular rheumatism is that form which affects single muscles or groups of muscles.Neuralgic rheumatism is a muscular rheumatism which is in reality a neuralgic affection of the sensory nerves.Nodular rheumatism is synonymous with arthritis deformans.Periosteal rheumatism is a variety of periostitis common in syphilitics.Puerperal rheumatism is a variety of the affection said to originate from some disorder of the urogenital tract in pregnant,lying-in or menstruating women,as does sometimes gonorrhoeal rheumatism.Scarlatinal rheumatism is an affection occurring in the course of scarlet fever occasionally,which may lead to suppurative arthritis. Scorbutic rheumatism is an enlargement of the joints in a person suffering from scurvy.Spinal rheumatism is a mild form of spinal meningitis.The term rheumatism of the spine is synonymous with spondylitis deformans.Rheumatism of the superior portion of the spinal cord is a synonym of tetany.Synovial rheumatism is a rheumatic affection of the synovial membranes usually leading to large serous effusions.Syphilitic rheumatism is synonymous with periosteal rheumatism.Rheumatism of the uterus is a true rheumatism of the uterine muscle,but,according to some authorities,a neuralgia of the uterus. Venereal rheumatism is synonymous with gonorrhoeal rheumatism.Vertebra-meningeal rheumatism is another name for spinal rheumatism.The term visceral rheumatism interprets itself. Coming now to the Latin equivalents,-rheumatismus calidus is a synonym of acute articular rheumatism. Rheumatismus cervicis is another name for torticollis. Rheumatismus cordis means rheumatism of the heart. Rheumatismus coxae is an optional name for coxalgia. Rheumatismus dorai is a rheumatism of the spinal muscles and ligaments. Rheumatismus febricosus is a synonym of rheumatic fever. Rheumatismus febrilis exanthematicus is an occasional designation of dengue. Rheumatismus flatuosus is a synonym of emphysema. Rheumatismus lumborum muscularis means lumbago.Rheumatismus nodosus designates rheumatoid arthritis.Rheumatismus odontalgicus is optional for odontalgia. Rheumatismus pectoralis is a synonym of pleurodynia.

PLAN OF NARRATIVE.

Obviously it is beyond the scope of this essay to enter into a full and critical account of all the above various conditions to which the term rheumatism has been in one sense or other applied. Such being,then, both unnecessary and inadvisable, I shall restrict my efforts to the consideration of such common forms of the disease as acute articular rheumatism(including the subacute variety and the affection in children), chronic rheumatism,muscular rheumatism,and gonorrhoeal rheumatism,as well as arthritis deformans.

A C U T E R H E U M A T I S M.

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S Y N O N Y M S.

Acute Articular Rheumatism; Rheumatic Fever; Acute Inflammatory Rheumatism; Acute Arthritic Fever; Acute Rheumatic Polyarthritis; Rheumarthritis; Polyarthritis Synovialis Acute; Rheumatismus Febricosus; Rheumatismus Calidus.

D E F I N I T I O N.

Acute articular rheumatism is a general, alleged infectious, febrile affection, attended with multiple inflammations, pre-eminently of the large joints, and very frequently of the heart, but also of many other organs - these inflammations observing no order in their invasion, succession or localisation, but when affecting the articulations tending to be temporary, erratic, and non-suppurating, when involving the internal organs proving more abiding, and often producing suppuration in serious membranes. It has been said to be connected with a diathesis, the arthritis, which may be inherited or acquired. It may present such modifications of its ordinary characters as to justify being called sometimes subacute articular rheumatism, and it may now and then pass into the chronic form. It is generally endemic, but occasionally an epidemicity has been observed.

The affection finds its initial expression in a sensation of chilliness or of rigor, and then follow a weakness and a condition of general out-of-sorts. Accompanying these phenomena are aching pains in various parts, and especially in the arms or legs. These soon increase in severity and are mainly complained of in the large joints, which rapidly become swollen and exquisitely tender to the touch. There is a coincident rise of temperature, and the skin is covered with a profuse, sour, and malodorous perspiration. The torturing pain in the joints is particularly noticeable and dominates the entire clinical picture of the disease. Such large joints as the knee, ankle, wrist, elbow, shoulder, and hip are generally alone involved, though the affection is sometimes observed in the knuckles, rarely in the joints of the foot where gout is usually seen. The affected articulations are painful, swollen, and remarkably sensitive to pressure or handling.

marked redness is not a common feature of these inflamed parts.The process exhibits a great tendency to migrate from joint to joint,and on several occasions during the evolution of the disease alternation of pain and comparative comfort may be experienced in most of the large joints of the body.The invasion of fresh joint is not invariably accompanied by subsidence of the inflammation in those already implicated,though now and then a true metastasis of the morbid process may be seen.The temperature is high and the pulse and respirations are increased in frequency.The hyperthermia is more irregular than continued,and the general phenomena exhibit considerable variation according to the individual case.The acidity of the copious and malodorous perspiration is marked,and the saliva may also have the same reaction.The urine is strikingly acid,of high specific gravity,copiously deposits urates,and has its urea-content greatly increased. The patient's bowels are constipated,the tongue is coated with a thick white fur,anorexia is present,and thirst is marked.The attitude of the patient is that of great helplessness,he lying on his back not daring to make the slightest movement lest his torture should be increased;even the streaming facial perspiration he dare not lift a hand to wipe away.He longs for relief to his sufferings,his expression is pitiable,and cannot tolerate the weight of the bedclothes or the presence of his ffiends.The affection lasts a variable time; left to itself,it seldom persists more more than a fortnight or three weeks;but now,under the orthodox treatment a dininished severity is always observed.Cardiac complications are common;and in this fact lies the great peril of the disease,as irreparable injury is generally inflicted on that delicate organ which sooner or later teminates the patient's life.Failing these cardiac troubles,the outlook is eminently favourable in properly managed cases.Malignant rheumatic cases are those in which the temperature runs up to a hyperpyrexial height,and is associated with severe nervous symptoms.

E T I O L O G Y.

HEREDITY.

A tendency to rheumatism is undoubtedly often transmitted by inheritance.The affection has occurred in the newborn,and the children of rheumatic progenitors are more liable to the disease than are others in in the proportion of five to one.The malady,like both gout and tuberculosis,may therefore be either directly transmitted through this agency,or more often a constitutional predisposition to its development seems to be inherited,in some cases as strikingly as in gout. That heredity predisposes to acute articular rheumatism is admitted by practically all modern authorities, even some of the older ones,whilst mentioning it as a traditional belief,not venturing to deny it.The

statistics of inheritance variously show that it is a factor in rheumatism in from twenty to fifty per cent. of cases.The frequency of the inherited predisposition Fuller placed at thirty-four per cent.;Beneke,quoted by Homolle(New Dict.of Med.& Surg.,T.xxxi,557),at a fraction over that figure;Pye-Smith(Guy's Hosp.Reps., S.3,xix,320)at twenty-three per cent.Most of the patients whom the statistics concern had but one rheumatic parent,but when there was a double inheritance,or the disease had been transmitted through several successive generations,it generally developed in a severe and recurrent or persistent form.Such predisposition favours the occurrence of the disease in early life,but does not necessarily determine an attack of acute rheumatism in the absence of other predisposing or exciting causes.That the inherited bias or mode of vital action or condition of tissue-health may be so great as,per se,to induce an attack of the disease,is held by some authors.It is probably that not only acute articular rheumatism in the parents,but simple chronic articular rheumatism and those forms grouped under the name of rheumatoid arthritis,may impart a predisposition to the acute as well as the chronic varieties of articular disease just mentioned.But owing to the obscurity which still surrounds the relations existing between acute articular rheumatoism and rheumatoid arthritis the point needs further investigation.In what the inherited predisposition to acute articular rheumatism consists is doubtful:to affirm that it imparts to the tissues or organs a disposition to react or act according to a fixed morbid type,or that some of the nutritive processes are perverted by it,is merely to vaunt a theory, not to explain the nature of the predisposition in question.A distinction should be drawn between the rheumatic diathesis and those exceptional instances in which the mother has suffered from acute articular rheumatism during pregnancy is the child is born with cardiac lesions.Here there has probably been a direct infection through the placental circulation.

AGE.

Qualified by the fact that rheumatism may occur at any time of life,it is,par excellence,an affection of early adult age,the largest number of cases occurring between fifteen and twenty-five years,and the next probably between twenty-five and thirty-five.A marked decline in its frequency takes place after the age of thirty-five,and a still greater after forty-five.It is not uncommon in children between ten and fifteen,but is very rare under five,although now and then one comes across a case of the disease in children three or four years of age.While the acute articular affections observed in sucklings are,as a general rule,either syphilitic or pyaemic,some authentic instances of rheumatic polyarthritis are recorded.Rauchfuss encountered only two cases of rheumatism amongst fifteen thousand infants at the breast during a period of four years. Widerhofer(Jahr.f.Kinderkr.,1859,157)saw the affection in an infant,aged twenty-three days,in the Foundling Hospital at Vienna,the only case of the kind that occurred among seventy thousand children in eight years. Staeger(Jour.f.Kinderkr.,1856,vi)met with it in a baby four weeks old;another case recorded by him is,to say the least of it,doubtful;and the same uncertainty clings to two out of the three cases reported by

Bouchut(Dis.of Childr.,1855,p.706).Henoch(Beit.z.Kinderh.,N.F.,1868)has recorded a case of acute rheumatism in a child of ten months;Roger(Arch.Gèn.,1867,i,p.54) describes its occurrence un a child of two,and another in one of three years.An analysis of the statistics of nearly five thousand(4908)cases before me,extended over a period of fifteen years,shows that under ten years there were 1.79 per cent.;from ten to fifteen, 8.1 per cent.;from fifteen to twenty-five,41.8 per cent.; from twenty-five to thirty-five,24.5 per cent.;from thirty-five to forty-five,14.2 per cent.;and above forty-five,9.4 per cent.Another statistical compilation covering ten years shows under fifteen years,4.38 per cent.;from fifteen to twenty-five,48.68 per cent.;from twenty-five to thirty-five,25.87 per cent.;from thirty-five to forty-five,13.6 per cent.;and above forty-five, 7.4 per cent.The close correspondence existing in the two tables for all the periods of life above fifteen is very striking:the disparity between them below the age of fifteen may,I think,be explained by the circumstance that the first tabulation covers places with large populations and numerous institutions.Doubtless these tables do not correctly represent the liability of children to acute articular rheumatism,but they are probably a fair statement of the relative frequency of the disease in the adult hospital populations of the localities concerned,London included.If primary attacks of the disease only were tabulated,the influence of youth would be more evident,for it is scarcely possible to find on record a clear and authentic instance of the disease showing itself for the first time after sixty. Pye-Smith(Guy's Hosp.Reps.,S.3,xix,317)has done this in 365 cases,and the results prove the great proclivity of very young persons to acute rheumatism:Between five and ten years 6 per cent.occurred;between eleven and twenty, 49 per cent.;from twenty-one to thirty,32.3 per cent.; from thirty-one to forty,9.5 per cent.;from forty-one to fifty,2.2 per cent.;and from fifty-one to sixty-one, 1.1 per cent.The same author has also shown that secondary attacks are most common in the young:so that advancing age not only renders a first attack of the disease improbable,but lessens the risk of of a recurrence of it;the influence of age upon acute rheumatism is further shown in the fact that the disease is less severe,and less apt to invade the heart,in elderly than in young individuals.Whipham(Brit.Med.Jour.,Feb.25,1888) has analysed a series of 655 cases of acute rheumatism.Of the whole number 80 per cent.occurred between the age of twenty and forty,only 32 having occurred in children under ten years.Of the whole 655 cases treated,22 died,chiefly from cardiac complications,and there occurred seventy relapses.There are numerous other statistical compilations in the literature showing also that the affection is sledom seen earlier than the tenth year,and,although numerous instances were formerly reported as developing in early infancy,many of them are now believed to have been due to scurvy with localised articular symptoms,produced by improper feeding with proprietary foods.Rotch(Pediatrics,p.1085)says that he has seen,however,general rheumatic polyarthritis in an infant two weeks old,and another case at the seventh month.Both patients recovered after several months' treatmentOf 8631 cases analysed by Besnier,

only 301 were found in childhood.Among 176 cases reported by Davaine and Lancereaux(Jour,de Méd.Interne,Vol. ii,No.3,p.75) the maximum frequency was found to be between the fifteenth and twenty-fifth years.Out of 1303 nalaysed by Fagge(Principles and Practice of Med.,Vol. ii,p.816)only 30 occurred as a first attack after the fiftieth year;later than this the subacute abd chronic forms may make their appearance,but first attacks of the acute type do not occur.The concenus of professional opinion is,then,in favour of the fact that the liability to the affection is greatest between the ages of fifteen and fifty.This immunity of the very young and very old can be accounted for only in one of two ways:either the rheumatic poison does not enter their systems,or having entered it does not act.The former is a position which cannot be maintained;for there is a no reason whatever why an agency which may gain entrance to the system at twenty or thirty years of age may not equally gain entrance at ten or sixty;the portals of the system are as free and as open at one age as at another.The second is a position which may reasonably be maintained,for there is distinct evidence that some poisons may,under certain circumstances,be introduced into the system without producing effect.We know,for instance,that a person who has not been vaccinated and has not had smallpox cannot be exposed to the poison of that disease without almost certainly taking it.But we also know that,having once suffered,he may be constantly exposed,and may even have the poison directly introduced into his system without again suffering from its action.So with each of the eruptive fevers:one attack confers,as a rule,immunity from the future action of its poison.Here we have ample proof that a poison,indeed a very potent one,may gain entrance to the patient's system without affecting it in any way.The virus of acute rheumatism operates upon the fibrous and serous tissues of the motor apparatus of the body,and its action is almost confined to the textures of those joints which enjoy a high degree of functional activity.But these textures are not equally active at all periods of life.In infancy and early life there does not exist the physical strength and stamina necessary for active movement.After the age of fourteen or fifteen the conditions are different;the child has now reached an age at which work and vigorous exercise begin to form part of his daily life;and when adult life is reached hard work and active exercise are of common occurrence.This implies the constant possibility of vigorous and free movements of the larger joints,and increased force and activity of the heart's action.It necessitates also a state of preparedness for such action.At any time a call may be made for efforts necessitating such movement and action;and the requisite facilitating and restraining forces must be there to meet the emergency.The period of life at which such efforts are made is fromfifteen to fifty,or thereabouts.This, therefore, represents the period of highest functional activity of those tissues whose duty it is to facilitate and restrain movement.It also represents their period of greatest liability to the action of the rheumatic poison.The period of liability to the action of the rheumatic poison corresponds exactly to the period of functional activity of the tissues specially involved in that disease.The same thing is noted in connection with

some of the eruptive fevers.It is specially marked in the case of typhoid fever.The intestinal glands whose inflammation constitutes the characteristic feature of that disease exist in infancy in but a rudimentary state.After two or three years they begin to increase in size and functional activity,and go on increasing till adult life is reached.From that time till middle age they are prominent objects in the intestinal wall.They then begin to diminish in size and functional activity and go on diminishing until in old age they are practically non-existent,and have ceased to exercise any function.The liability to the action of typhoid poisons is directly as the size and functional activity of these glands.In infancy and old age the disease is rare.The period of greatest liability to it is from fifteen to thirty-five or thereabouts.All this is adequately explained on the grounds that these glands are the nidus of the typhoid poison and are essential to its propagation in the body of the patient.It is similarly that the special tendency of the rheumatic poison to affect persons between the ages mentioned can be explained.This is the period of functional cativity of those textures whose inflammation constitutes the special lesion of the disease.They form a suitable nidus for the propagation of the rheumatic poison only during their period of functional activity;it is during that period,then,that individuals ate most likely to suffer from the disease.

SEX.

Most authorities assert that sex exercises no direct influence beyond exposing males more than females to some of the predisposing and exciting causes of acute rheumatism;this general opinion is perhaps true of the statement be confined to adults,to whom,indeed, most of the available statistics apply.But it should be remembered that a larger proportion of men than of women report to hospitals;and there is some reason to believe that in childhood the greater liability to the disease is on the part of the female sex.Thus,according to Tuckwell(Contributions to the Pathology of Chorea,St. Barth.Hosp.Reps.,v,102),the number of cases treated at the Children's Hospital in London from 1852 to 1868 was 478,in whom rheumatism was seen in 226 males and 252 females.Of Goodhart's 44 cases of acute rheumatism in children 26 were girls and 18 were boys(Guy's Hosp. Reps.,S.3,xxv,106).Of 57 examples of rheumatism in connection with chorea observed by Roger(Arch.Gen.,Vol.ii, 641,1866,and Vol.i,54,1867)in children under fourteen, 24 were males and 33 were females.Wunderlich had 65 males to 43 females,and among 230 cases Lebert found 119 males and 111 females.Roth had 38 males to 41 females;Fiedler 281 males to 370 females,in another year 61 males to 64 females;Kreuser(Würt.Corresp.Blatt., 1866,No.2),in Stuttgart,44 males to 43 females;Huber 49 males to 41 females.In the Rudolfsspital at Vienna, in 1871,they had 75 males to 45 feamles;in 1872,62 males to 46 females,admitted with acute rheumatism.My own experience had been that in early life females are more prone to the disease than males,especially at the period of puberty,but that after the twentieth year, when the influences of occupation,exposure,and hardship predominate,males are much more often affected;taking all ages into account,about one third more cases have occurred in my practice amongst males than females.

CLIMATE AND SEASON.

It is usually held that acute articular rheumatism belong especially to temperate climates, and that it is extremelyrare in the polar regions; but respecting its prevalence in the tropics extremely contradictory statements are made. Vel declares that it is not a disease of hot climates; Levison saw only 4 cases of acute articular rheumatism, and not one of chorea, in Martinique during twenty years' practice; while Bey says it is common in Egypt, and Webb remarks the same for the East Indies. Even in temperate climates, like those of the Isle of Wight, Guernsey, Cornwall, some parts of Belgium, the affection is rare, a circumstance regarding which there has been considerable disputation. Though the disease is apparently more common in cold, damp climates than elsewhere, it is not infrequently encountered in such climates as that of California, the being seldom seenin the tropics unless recently imported there. While tropical and subtropical countries have no absolute immunity from rheumatism, and some of them, such as Centtral Arabia, India, the Cape, the Abyssinian plateua and Peru, are much subject to it nowadays, yet the maximum of frequency falls quite decidedly within the higher latitudes. The disease occurs at all seasons of the year, although subject to moderate variations dependent mainly upon atmospheric conditions. It is the general opinion that it prevails most during the cold and variable months of spring, but this is not true of every place, nor invariably of the same place. Indeed, Besnier (Encyclopaedic Dictionary of Medical Sciences, S.3, Vol.iv), after a long and special observation of the disease in Paris, concludes that there it is most frequent in summer and in spring. The statistics of Copenhagen, Berlin, and Zurich show a minimum prevalence in summer or in summer and autumn. Sudden changes of weather are particularly unfavourable, but have more influence upon the chronic than the acute form. Probably for such reasons as these rheumatism is more often developed in this country in the late autumn and early spring: seasons which are productive of unsettled temperature and melting snow, and rain. Sudden variations in the weather produce more effect upon chronic than acute articular rheumatism. The majority of my cases have occurred in autumn and spring; the fewest have occurred in summer; but I have seen them all the year round. I have seen the affection more often in urban than rural localities, in the former of which it is favoured by cold and damp. Newsholme says that it has to do with a low level of the ground-water; in London, it is true, the disease is more prevalent and more severe when the cold and rain set in after a hot and dry summer

SOIL.

The earlier authorities on rheumatism endeavoured to refer the frequent development of the disease to the nature of the soil, damp and mouldy soils being thought most injurious; but this belief has been modified by the infection-theory hereinafter to be described. Longstaff has found the disease more common where the soil is dry, and Newsholme, as stated, mentions the fact that a low subsoil water with high temperatures favours the breeding of many germs in the soil. As the affection originates in so many different surroundings, it is doubtful whether the question of the soil has much, if anything, to do with it. Damp alone does not explain the

frequency of the disease,if,as seems to be the case,it is a fact that it is an urban disease and more severe when the subsoil water is low.Only in so far as they affect the climate of a locality may we take into etiological consideration the peculiarities of the soil,such as elevation,configuration,the kind of rock,and the physical characters.It is in that sense that we have to interpret the well-known preference of the disease for open basins and plateaus exposed to the wind,for damp and deeply cleft valleys,and for sea coasts or the shores of great rivers.There are,unfortunately,no statistical materials for instituting a comparison of the affection on the different kind of soils.Only slight differences appear from the statistics of Balfour regarding the prevalence of the disease amongst British and native troops quartered at various elevations in the Madras Presidency.Amongst the British troops on coast stations there were 174 cases;at stations on the plains, 113 cases;and on elevated stations,126 cases.Amongst the native soldiers these figures were respectively 57, 58, and 52. It seems to me thatmthe evidence brought forward in favour of the production of the disease through the direct influence of a wet or marshy soil rests on unreliable observations.

ELIMINATORY DEFECTS.

Checking of the secretions,should it take place suddenly,of the kidneys or skin,or of the excreta from the bowels from any cause,by determining accumulation of waste products in the blood,constitutes a strong influence;in short we may regard as pernicious anything which renders the blood less solvent and restricts elimination and free oxidation.

COLD.

Not a few authorities hold that residence in damp and cold houses predisposes somewhat to acute articular rheumatism,although not at all to the same degree that it does to the chronic articular and muscular forms. Chomel and Jaccoud especially have insisted that it will gradually create a predisposition to the disease, even if it has not been inherited.Innumerable others agree that cold is the most frequent exciting cause of the disease,and that it is especially effective when applied to the body while it is perspiring freely or is overheated or fatigued by exercise.There is no necessary ratio between the degree of cold and its duration and the severity of the resulting rheumatism.A slight chilling or a momentary exposure to a current of cold air will in some as as powerfully and as certainly as a prolonged immersion in cold water or a night spent in sleeping on the damp grass.This circumstance,together with the fact that cold applied in the same way may also produce a pharyngitis or a bronchitis,a pneumonia or a nephritis,etc.,is held to indicate that the cold acts according to individual predisposition;and Jaccoud, Flint,and others maintain that unless a rheumatic proclivity exists cold will not produce an attack of the disease under consideration.I doubt that we are yet in a position to assert that absolutely,although the weight of argument is in its favour.Let it suffice to say,that while a prolonged residence in a cold,damp dwelling may gradually develop a predisposition to rheumatism,a short exposure to cold will be unlikely to induce an attack of acute articular rheumatism is the patient is pre-

disposed to it.In short,exposure to cold never in itself causes rheumatic fever,but may precipitate an attack when additional influences prevail;under such conditions an individual may develop the affection through getting wet and chilled in the rain,perspiring heavily and sitting in a draught,or falling overboard.

GEOGRAPHICAL CONDITIONS.

Acute articular rheumatism, we have seen,is more often encountered in cold and dampt regions than elsewhere.It is one of the commonest affections of this country.In the year 1888 its prevalence here was inquired into by a Collective Investigation Committee of the British Medical Association,who found that there was no part of the Briths Islands free from an extensive prevalence of the disease.Referring to London, Macelod(London Medical Gaz.,Oct.,1837)says that "rheumatic fever is a disease exceedingly prevalent among the class of persons admitted into the hospitals of this metropolis;indeed,so true is this,that I am satisfied all medical men connected with those institutions will bear me out in the assertion that,if we take into consideration the ulterior effects of acute rheumatism,it is not only one of the most prevalent,but one of the most fatal maladies incident to our precarious climate." Omerod(Med.Times & Gaz.,1852,523)later confirmed this with the statement that the number of patients treated for it in the London Hospitals reaches the enormous figure of 11.5 per cent.of all the admissions.As a rule, it is common in the midland and northern counties than in the south,as would appear from the statistical return from Stockport(Watson,-Trans.Prov.Med.Assoc.,1834,ii, 196),Bristol(Symonds),Manchester(Lyon,- Brit.Med.Jour., Dec.,1870,597),and other places.Dealing with Cornwall, Forbes(Trans.Prov.Med.Assoc.,1836,iv,174)says that "the whole cases seen by me in four years and a half were four in number,viz.,two in 1871 and two in 1821,not one case having been met with in the three intermediate years.In the dispensary report for 1821 I stated:during the whole of the last two years,not one case of acute rheumatism has been entered in the books,and I cannot help thinking that the total absence of the disease,among so large a body of individuals,for so long a period,is a circumstance that would be reckoned very singular in the northern or even central part of our island!"The same immunity is reported of Devonshire by Jefferey(Ibid.,1843,xi,226),Shapter,and others;and also of the Isle of Wight and Guernsey by Hoskins(Lond.Jour. of Med.,Aug.,1852,as well as others subsequently,although the affection isby no means infrequently observed amongst sailors and other living on the coast.In France there are no statistical records of the prevalence of acute rheumatism;but it would appear from accounts now and then published that the malady is common enough throughout the whole of that country,especially in the central and northern parts.The same remark applies pretty much to Belgium also.In Germany acute articular rheumatism and cardiac disease are common enough in Bavaria and in Alsace-Lorraine;this has been out down to the enormous quantities of beer consumed there.The disease is just as common in Swabia as Bavaria.The malady is often seen in Denmark,where,according to Newsholme,it varies slightly in amount from year to year;in recent times its maximum prevalence has been in

1871,1873,and 1883-1888.The fatalities arising from this affection in the towns of Denmark between 1892 and 1897 varied from 37 in the first to 55 in the latter year.The prevalence of the disease is much about the same in Norway,where the years of maximum prevalence were respectively 1871,1876,and 1888-89.The malady is also by no means rare in Sweden,particularly in the northern regions of the country.The returns from the far-north regions negative the statement that acute articular rheumatism is almost unknown in such localities.In Kamschatka,according to Bogorodsky,the affection is common enough;in Iceland Finsen saw 20 cases during ten years in a district with a population of ten thousand;in the Faröe Islands Panum had several cases to treat during a comparatively short residence; and Lange says that it is by no means unknown in Greenland.There have been large numbers of cases in the northern and temperate latitudes of the Eastern Hemisphere,but the data are too incomplete for definite conclusions as to relative frequency to be derived.Per hundred of all admissions to hospital there were in Christiania Reichshospital(Nord.Mag.f.Laegev.),during seven years,3.5.In Copenhagen Frederisshospital,in twenty-four years,4.0.In the Rostock Town Hospital,in two years,3.3(Fiedler,-Arch.der Helik.,1866,vii,157). In the Bremen Town Hospital(Barkhausen,- Arch.f.d.Ges. Med.,1841,i,332),during twelve years,2.0.In the Hamburg General Hospital(Tungel,- Klin.Mitth.),in three years, 4.5.In the Dresden General Hospital(Fiedler,- Loc.cit.), during thirteen years,4.5.In the Giessen Clinical Institute(Roth,- Würt.med.Zeit.,1863),during a similar period,5.0.In the Würtzburg Julius-Spital(Ibid.),in four years,1.5.In Stuttgart Katherinen-Hospital(Mitth.des Würt.aerztl.Vereins,1834,114,340;Würt.aerztl.Corresp., Jahrg.,iii),during twenty-four years,4.5.In the Town Hospital of Zurich(Lebert,- Handb.der pract.Med.Tübing., 1859,ii,853),during a similar period,4.5.Russian statistical return are notoriously defective;but it would appear that acute articular rheumatism is very common along the shores of the Gulf of Finland,and elsewhere, Siberia and the Caucasus included.One cannot compare its frequency in Russia with that of other countries,as the affection is not treated separately from other fevers in the official returns.The disease is common enough on the Bavarian plateau,where it has the foremost place amongst the acute diseases as regards the number of cases(Annal.der Städt.Allg.Krankenh.z.München,1878, i).It is by no means a stranger to various mountainous part of the Canton Vaud,where there is more rheumatic fever than in other Swiss Cantons(Harpe,- Schweiz.Zeit. f.Med.,1849,157).The same is true regarding the prevalence of the affection in Austria-Hungary.In Italy acute articular rheumatism,from 1887 to 1896,had a mortality varying from 25 to 36 per million inhabitants;and during the same period the chronic form of the malady was even more fatal;in such parts of the country as Turin,Genoa,Alessandria,and Mantua rheumatic fever is regarded as one of the commonest of ordinary diseases. In Spain and Portugal there is a lot of rheumatism;it appears to be endemic on the plateau of Castile and in the Asturias,and in Seville,Granada,Valencia,and Malaga it is very commonly observed.The people of the Balkan peninsula are very frequently rheumatic;and in the Salonika province of Turkey and elsewhere it is often

seen;it appears to be more common in the mountains than in the plains.In the highlands of Montenegro rheumatic fever is very prevalent,and this is ascribed to the severity and dampness of the climate.No disease is more common than this in Constantinople and along the shores of the Bosphorus and the Sea of Marmora;and elsewhere in that country the malady is very well known.In some regions of Asia Minor acute articular rheumatism is very common.It is often encountered in Trebizond,due, it is said,to the excessive moisture in the air from the Black Sea on which it lies.The affection has been long known in Arabia(Lond.Jour.of Med.,Aug.,1852);and it is very common in Mitylene and some other islands on the seashore,as well as at Tripoli on the coast of Syria.The Caucasian populations are very well acquainted with rheumatic fever.In central Asiatic Russia,as at Turkestan and Transcaspia,it appears to be much less prevalent than in European Russia,though this statement is probably based on incomplete returns.Acute articular rheumatism is very often encountered in all parts of Siberia,no part of the country being immune;it is said, however,to be a rare affection in Kamschatka and Tomsk. It is a common disease in Afghanistan and Persia,The poorer classes and those exposed to all sorts of rigorous weather at Khanikin upon the Turco-Persian frontier, after the heavy rains of early winter,suffer considerably from this disease.Rheumatic fever is by no means unknown in India,though the statements regarding that country have been from time to time rather conflicting. Some authors have dwelt on the rarity of all forms of rheumatism in India,including cardiac complications. Thus Buchanan(Jour.Trop.Med.,Nov.,1899)writes that not only acute rheumatism but also chronic rheumatism and organic cardiac murmurs are seldom encountered there; and it seems that Cantlie(Ibid.,Oct.,1899)on his tour of Indian cities never met with a case in the hospital he visited.Some of the earlier writers,such as Malcolmson and McGregor speak of it as rare,and others,such as Shanks,Macpherson,Morehead,Gordon,Day and Curran,conclude from their own experience that it is almost confined to Europeans,and is very much milder than in Europe,the cardiac complications in particular being seen only now and then.On the other hand,Parry(Madras Quart.Med.Jour., April,1842,143)says that he is inclined to believe that organic lesions of the heat originating from acute articular rheumatism are much more prevalent affections and a more frequent source of inefficiency amongst our troops in India than is generally supposed;and a similar opinion is given by Wallace,Evans,Geddes,Winchester, Huillet,and others.But perhaps the most decisive statement is that of Webb(Pathologia Indica,Lond.,1848,p. 77),who remarks in connection with the numerous preparations of pericarditis and endocarditis in the pathological museum of the Calcutta Medical College:"Now can we wonder at this;for what disease in India is more universally diffused over the country than articular rheumatism,and what sequences more common to it than endocarditis and pericarditis?"He mentions also the fact that he has seen cases of rheumatic fever all over India. The official returns would seem to indicate a common prevalence of the disease nowadays amongst both natives and Europeans;and in the British Army in India there are more cases of rheumatic fever and deaths from it

than in England.According to Davidson,Bengal is the Presidency in which the disease is most common,Bombay that in which it is most fatal,and Madras that in which there are fewer admission and fatalities.It is somewhat uncommon in Rajputana,though other forms of the disease are by no means infrequently seen.The affection is just as rare in Assam and in the Malay Peninsula,and few,if any cases have been reported in Penang and Singapore. McClosky(Jour.Trop.Med.,Jan.,1900)says that,during the eight years he practised in the Straits Settlements and the Malay Peninsula,he never saw rheumatic fever occur in a native,and only once in a white man who had suffered from the disease prior to leaving home;cardiac complications are quite rare,and only at the autopsy is the presence of such observed.Most of the people of the East Indian Islands are liable to attacks of the disease,though perhaps less so than others elsewhere. The disease is common in Java and in British North Borneo;in Summatra and the Lampongs it is widely prevalent,as also in the Rhio archipelago.The affection is rare or somewhat mild in type in the Dutch East Indies, both amongst the white and coloured peoples.It is difficult to estimate the prevalence of rheumatic fever in China;Cantlie(Jour.Trop.Med.,Oct.,1899)says that he never saw a case during the many years of his hospital practice in that country.The medical officers in the service of the Chinese customs frequentlymentions its occurrence(Chinese Customs Med.Reps.,No.52);thus,in 1895-96,15 cases of rheumatic fever were recorded at Lungchow.The affection is one of the commonest at Ichang;and Coltman(The Chinese,Their Present,etc.,1891) informs us that the malady is common in Foochow,Shanghai,Soochow,Chifu,Lao Ling,Hangchow,and other parts of China.The Chinese inhabitants in Formosa seem to be much troubled with this ailment.In Mongolia it is one of the commonest diseases.Acute articular rheumatism would seem to be very rarely seen in Japan,particularly in Yokohama and Tokio.In the latter city Scheube(Virchow's Arch.,1885,Bd.99,377)saw only 39 cases in four years(7 of these in hospital) and 4 with cardiac complications.Wernich(loc.cit.)likewise gives the disease as of rare occurrence in that country,and of a milder type than in Europe.Both the natives and foreigners in British New Guinea suffer now and then from rheumatism. In Australia it is a rare disease,or at least is not nearly so common as in out own country.Seldom is the affection mentioned in the official statistics of the different colonies,although in South Australia and New South Wales the number of deaths from rheumatic fever is rather large,while cardiac complications are frequently seen.The malady is rare in New Zealand,though quite the contrary is true regarding many of the islands of the Pacific,particularly New Caledonia,the Society Island,the Marquesas group,and the Sandwich Islands.Formerly the affection was seldom seen in Samoa:now,however,it is by no means infrequently enoountered.In Madagascar rheumatic fever is of rare occurrence amongst the natives,though some of the other varieties of rheumatism are the reverse of this. Zanzibar(Christie,- Brit.Med.Jour.,June,1872)is said to enjoy a complete immunity from the disease,Christie not seeing a single case during five years' observation. Borius,however,says he has seen several cases of it

on the small island of St.Marie.According to Davidson, the affection is very common amongst both the matives and Europeans in Tunis,where it constitutes a third of all the known diseases.On the other hand,it is seldom seen in Algeria and Morocco.It has been seen comparatively often in the Cape Verde Islands,in Nubia,and on the Abyssinian plateau;in Egypt,too,it seems now to be much more common than in former years.The malady is one of the commonest seen in Senegal on the West Coast of Africa;in Gambia it is well known;in Sierra Leone and Lagos it is an everyday disorder;and on the Gold Coast the natives very often fall victims to its effects. Now and then a case is seen in the Cameroons;and it is very commonly encountered in the Congo Free State.Pruen says that rheumatic fever is seldom seen in Central Africa,though chronic rheumatisn often is,and cardiac complications also.In Uganda there is plenty of acute articular rheumatism,and in Manyuema it very often kills the natives.One of its worst seats on African soil is at the Cape,according to the experience of every authority,it being particularly common in Transkei, Trembuland,and Pondoland,as well as Bechuanaland;on the other hand,it is seldom seen in Mashonaland,where, according to the colonial reports,it is the rarest of diseases;Davidson,however,says that the affection is now and then seen in the Seychelles.I have it on reliable authority that a large proportion of the hospital cases in Cape Colony and Natal are rheumatism in its acute or other forms.In Mauritius and Rodriguez the malady is common enough.Coming now to North America, it appears that the disease is known in Greenland,and also over the whole of Canada.In the United States the south is said to be more rheumatic than the north;and in Massachussetts the rheumatic mortality has much increased of late.Osler says that ruematism is more prevalent in Montreal than in Philadelphia and Baltimore. Generally speaking,it is difficult to judge of the area and frequency of rheumatic fever in North America,owing to the extreme scantiness of available data;but if we may judge from the number of communications made about it to medical societies,it should be as common on the whole as it is in the temperate latitudes of the rest of the world.In Mexico,on the Anhuac plateau,it is of frequent occurrence;it is extremely rare in the low-lyingcoast town of Vera Cruz and others on the littoral;at all events Heinemann says that he never saw a case of rheumatic fever during the several years he practised in these parts.Acute articular rheumatism is often seen in the West Indies,where it is common in Jamaica,Grenada,St.Vincent,Antigua,and St.Lucia.In the Virgin Island it is a very common disease,and in the Leeward Islands and the Bahamas it is very often seen. In the latter,during the years 1897 and 1898 hospital admissions from it were strikingly great.Data regarding Central America are difficult to obtain;but it would seem that the affection is rare in Guatemala,and almost unknown in British Honduras.The same comparative immunity holds for British Guiana and French Guiana, where other forms of rheumatic affections are commonly observed.Acute articular rheumatism is by no means rare in Brazil and in the Argentine Republic;both in the capital and in the interior,along the Parana, Uruguay,and Paraguay rivers it frequently prevails.

Ecuador, Chile, Peru, and along the western littoral of the South American continent would seem to be hot-beds of the disease, and it quite an endemic malady of the River Plate States.

TRAUMATISM.

Injuries to the articulations, such as sprains, blows, falls, etc., while not directly causative, are said to often localise the inflammatory symptoms in the particular joints affected, and so induce an attack of rheumatism. A blow on the finger, the extraction of a tooth, a hypodermic injection, etc., may act powerfully in some persons uponand through the nervous system, and by lessening their resisting power may favour the overt manifestation of the rheumatic predisposition. But doubtless some such cases have been examples of mere coincidence. Unusual fatigue or overwork of certain groups of muscles and joints may have like influence. Repeated injuries of rheumatic joints may cause the affection to develop into the chronic form.

SOCIAL CONDITION.

Rheumatic fever may be seen in individuals of any social class; but it is less often encountered amongst the affluent than amongst those who are poorly clad, ill-fed, and who live in damp, dark, unhygienic surroundings. Such are influences that may be considered the auxiliaries to cold in exciting an attack, as they seem to increase the susceptibility of the patient to its operation: they establish what has been felicitously called a state of modbif opportunity. Such as all influences that reduce the resisting powers of the organs and organism, particularly bodily fatigue, mental exhaustion, the depressing passions, excessive venery, prolonged lactation, loss of blood, etc.

OCCUPATION.

It would seem that certain occupations predispose to the occurrence of rheumatism, notably those requiring prolonged exposure to cold and wet, sudden and extreme changes of temperature, especially during active bodily exertion and muscular fatigue. Thus it prevails amongst cabmen, drivers, stokers, sailors, fishermen, 'longshoremen, blacksmiths, laundresses, bakers, soldiers, cooks, maidservants, charwomen, and labourers generally.

DIET.

Most authorities hold that diet is without marked influence upon the incidence of the disease; and excess of meat or carbohydrate in the food is not now given the etiological interpretation of former times. Neither can an excess of urice acid in the blood be considered responsible for its causation.

ASSOCIATED DISEASES.

It is a well-known fact that there are certain pathological, and even physiological, conditions during or after which an inflammatory affection of one or several joints, closely resembling acute articular rheumatism, more or less frequently arises. Thus, during the early desquamating stage of scarlet fever a mild inflammation of the joints of the hands and feet, and frequently of the large articulations as well, is very often seen; and it is attended with profuse perspiration, with a condition of the urine like that of ordinary acute rheumatism, and occasionally with inflammation of the heart or pleura. During convalescence from dysentery an affection of a single or of several articulations

resembling rheumatism has been noticed,and the two affections have even alternated in the sam patient. That singular epidemic disease,dengue,is attended with a polyarticular affection closely resembling acute articular rheumatism,occasionally pursuing a protracted course,and not seldom leaving after it a cardiac lesion. In haemophilia polyarticular and muscular disorders frequently arise which closely resemble,and appear to be sometimes identical with,ordimary adute articular and muscular rheumatism.Gonorrhoea,too,is often associated with a febrile polyarthritis,and rarely with an endocarditis at the same time.In the puerperal state an inflammation of one or several articulations is not rarely seen,and to it the term puerperal rheumatism,as we have seen,has been applied.Malaria is not infreuently associated with rheumatism,and it is quite possible for gout and the latter to occur in the same person,although this combination is much less common than is ppularly believed.Severe nervous shock,exhaustion,general debility,starvation,chronic alcoholism,and anaemia may all be regarded as predisposing influences.Diphtheria and mumps may be accompanied by rheumatic symptoms,and chronic endocarditis is regarded as a predisposing factor.In a series of 56 cases of goitre reported by West there was a history of rheumatism in eleven per cent. Everything is not yet known regarding the real nature of the above-mentioned polyarticular inflammations;whist many of them are of a pyaemic nature,as some examples of puerperal and scarlatinal arhtirits,in which pus form-s in or about the joints and in the serous cavities and viscera,some of them are doubtless examples of genuine rheumatism occurring in persons of rheumatic predisposition,which have either been induced by the lowering influence of the disease upon which they have supervened,or by the accidental coincidence of some of the other causes of acute articular rheumatism.There remains,however,the ordinary form of scarlatinal nephritis,which so closely resembles true acute articular rheumatism in its symptoms,course,visceral complications,and morbid anatomy that it cannot be said that the two affections are distinct and different.And much the same appears to be true of the articular affections of dengue.Yet so frequently does the articular affection accompany scarlatina and dengue respectively that it cannot logically be referred to a coexisting rheumatic predisposition,and must be in consequence a consequence of the disturbing influences of the specific poison of those zymotic maladies itself.

TEMPERAMENT.

There is no valid reason to believe that any kind of bodily conformation or temperament has to do with a tendency to acute articular rheumatism,nor is there any change in the constitution of the tissues or fluids of the body by which the proclivity may be recognised. We infer the existence of the inherited predisposition,-the innate bias,-when rheumatism is found in the family history;when acute rheumatism or heart disease,or chorea not produced by mental causes,occurs in childhood;when the first attack of acute articular rheumatism is succeeded by subsequent attacks;and especially when the intervals between the attacks are short.Goodhardt has furnished valuable,but not conclusive,evidence to prove that in children obstinate headaches,night-terrors,

severe anaemia,various neuro-muscular derangements,such as torticollis,tetany,muscular tremors,stammering, incontinence of urine,recurring attacks of abdominal pain, with looseness of the bowels very soon after a meal,the cutaneous affection known as erythema nodosum,are indic-ations of a rheumatic bias,predisposition,or diathesis. (Guy's Hosp.Reps.,S.3,xxv).

P A T H O L O G Y.

GENERAL CONSIDERATIONS.

The pathology of acute articular rheumatism is a very much debated question,and is not at all satisfactorily known;indeed,to the minds of some it appears even more obscure than that of gout.Naturally,a disease at once so prevalent and so severe has stimulated research as to its cause and preventive treatment,and many able arguments have been advanced in support of conflicting theories.The fundamental question is whether or no the affection should be regarded as an active manifestation of a general arthritic diathesis,that is, a constitutional tendency,either hereditary or acquired, to the localisation of disease in articular structures. Widal says that the arthritic diathesis is to rheumatism what the scrofulous diathesis is to tuberculosis. Haig,who has contributed many years of study and a large clinical experience,believes in such a diathesis or common dyscrasia as the starting-point from which in one individual gout may develop,in another rheumatism,in a third arthritis deformans.Other clinicians maintain with equal emphasis that rheumatism is a disease of independent origin,possessing no pathological affiliation with either gout or arthritis deformans,and having nothing in common with this disease beyond the clinical fact that in all three the joints are the chief sufferers.Of late years there has been a decided tendency to forsake the doctrine of diathesis in general, which has been largely fostered by a wider knowledge of the pathology of the blood and of the toxaemias of infections.A good illustration of this fact is found in the modern view of the infectiousness of tuberculosis as of far more importance than a supposed tubercular or scrofulous hereditary diathesis.Authorities everywhere are agreed that the lesions of rheumatism are of an inflammatory nature,but differences appear in ideas when the nature of the inflammatory process in question is discussed.Some authors look on it as differing from ordinary inflammation only in its particular location; whereas others hold that it is specific in nature,and resulting from a special poison which does not operate in the production of other than rheumatic inflammation. The former is the view taken by those who consider the affection as the direct result of exposure to cold and damp;the latter that held by those who regard it as due to the action of a specific pathogenic germ circulating in the blood-stream.That exposure to cold and damp suffices to produce acute rheumatism is an old view which finds its chief support in the fact that the malady often occurs after such exposure.But so often does such exposure occur that it would be difficult

to mention any affection which might not be attributed to it in some way or other.Thus,if rheumatic fever is due to exposure to cold and damp,it should be most common in the coldest climates,and during the coldest weather. But it is a disease of temperate climates,not of the polar regions;and it is not always most often observed in the winter,even in temperate zones.Again,if cold be the cause,it should be most common in children and aged individuals who have little power of resisting the effects of low temperature.But just the opposite is the case,and the affection is most often seen from fifteen to fifty,that is to say,at the very time of life when cold can best be resisted.If caused by cold,the joints which suffer most from such exposure,those of the fingers and toes,should also suffer more from theumatism; but theyare very seldom involved in the disease.Furthermore,if this be the cause of the affection,it is puzzling to understand how pericarditis so often occurs and pleurisy and peritonitis so seldom.The pleura and peritoneum are just as much exposed to cold as the pericardium,and probably more so.The occurrence of endocarditis,and the almost entire limitation of this to the left side of the heart,cannot be explained on the temperature theory.It is a well-known fact that during the evolution of the disease fresh joints may be attacked after the patient has been confined to bed in a warm room for days or perhaps weeks.These later articular attacks are identical in nature with the earlier ones which ushered in the illness,and it would be unreasonable not to regard them as produced in the same way,and as due to the same cause.It is absurd to say that they are produced by cold.On similar grounds the early and primary articulars affections cannot be due to cold-exposure,against which also,not a few other obstacles can be advanced. The tendency to rheumatism is inherited;the disease is specially apt to occur at from fifteen to fifty years of age;it may attack the same individual several times; it does not confine its operations to one joint,but simultaneously or successively may involve several;it attacks also the membranes of the heart;it seldom proceeds to suppuration;and it is not much benefited by simple antiphlogistic remedies,but is promptly checked by measures which aim at the vanguishment of the constitutional disease.These facts show that no external agency and no amount of exposure can produce the malady, and the etiological factor must be of internal location. The allged local nature of the disease is negatived by the fact of hereditary transmission,a peculiar diathesis or condition of the system being handed down from parent to offspring.This inheritance can take place only in connection with constitutional affections,and not such local lesions as pleuritic,peritonitic,nephritic,and other inflammations.Rheumatism and other constitutional maladies are characterised also by their tendency to attack individuals of a certain age;and the idea of a constitutional predisposition is still further favoured by the liability to repeated attacks in the same person. The presence of a generally-operating internal and constitutional cause is suggested by the fact that many joints suffer at the same time or one after the other, which could not be the case were the affection purely local and external.The existence of the cause within the system alone will satisfactorily explain the

occurrence of hear affections in this disease;and that rheumatic is essentially different from all other forms of inflammation is suggested by the rarity of suppuration,even in severe and protracted cases.One often sees the worst forms of rheumatic fever promptly checked by the administration of the salicylates,which would not be the case with cerebritis,pneumonia,or hepatitis,the articular affections seeming to stand still until it is exhibited.The success of constitutional,and the futility of local,treatment complete the establishment of the fact that the affection takes origin within the system and never from a local or external cause.Nevertheless,exposure to such influences as cold and damp will sometimes act to determine an attack of rheumatic fever,though by no means in themselves giving rise to the disease.It will facilitate the study of this phase of the subject of acute articular rheumatism to subdivide the hypotheses concerning its origin under the headings that will now appear:

LACTIC-ACID THEORY.

In view of our present knowledge regarding this disease,no one ingredient of the blood,accidental or otherwise,and not excepting lactic and uric acids, can be defined as alone responsible for the arthritic changes of acute rheumatism.In gout uric acid is held responsible for the articular lesions;but the theory, that lactic acid fulfilled a like rôle for rheumatism, is no longer tenable.The failure of the exclusively alkaline treatment did much to discredit this theory. Noxious principles which circulate within the body are either manufactured within it or enter it from the outside.The rheumatic poison was assigned to the former category and regarded as the outcome of mal-assimilation or imperfect tissue-change.The occurrence of hyperacidity,as manifested in the hyperacidity of the copious sweats and hyperacid urine was the starting-point for the theory that an acid condition of the blood was productive of the clinical phenomena in this disease.Prout was the first to assert that the noxious agent was lactic acid,and that the symptoms of the affection directly resulted from the accumulation of this acid in the blood.This idea was enthusiastically accepted by others, who regarded the hypothesis as the best explanation of the symptom-complex hitherto advanced,the more so as it gave a clue to the cure of the affection by the alkaline method.But there soon arose not a few objectors to this apparently plausible line of argumentation,the failure of the alkaline treatment being strongly urged against it.Lactic acid is a product of tissue metamorphosis.It is an unstable compound,which readily undergoes change,and is excreted by the lungs and skin in the form of carbonic acid and water.It accumulates in the system under conditions of defective elimination or increased formation,or when these two factors act in conjunction with one another,the latter idea being the apotheosis of the idea.The acid is formed in the course of the metabolism of the muscles.During exercise it is formed in larger quantity than during rest,and when exercise ceases there is an excess of this acid in the system.But the exercise which causes increased formation of lactic acid is accompanied also by increased action of the lungs and skin,the channels by which the acid is eliminated in the form of carbonic acid and

water.Excessive formation is thus counterbalanced by increased elimination,and no accumulation takes place. But if,at this time,the action of the skin be checked, the metamorphosis and elimination of the acid are arrested,it accumulates in the system,and the clinical phenomena of rheumatic fever are observed.A chilling of the body surface would checked the elimination in question.The advocates of this eminently convenient theory particularly insisted upon the facts that rheumatic fever is accompanied by excess of lactic acid in the system,the disease never occurring without such excess, which excess is never found in any other disease;the injection of lactic acid into lower animals is followed by articular lesions such as occur in this disease; and the administration of the acid to human beings will produce phenomena closely resembling those of the same affection.Now to consider these pillars of argument in detail,there is no doubt that rheumatic fever is accompanied by excess of lactic acid in the system,and that such excess is noted only in connection with it. The important point is the relationship which this excess of acid bears to the pathological changes in the disease: that is to say,we must find out of lactic acid is the rheumatic poison,if its presence in excess in the system causes the clinical phenomena,or if the excess of the acid is,like pain and pyrexia,merely one of the features of the disease as produced by some other agent. Experience has shown that there are good reasons for refusing to subscribe to the theory of acid causation. Were the contrary to obtain,the rheumatic symptoms should last so long as the acid existed in adequate excess in the system,and should decline when it ceased to do so,and not till then.Remedies,too,which neutralise the acid should also cure the disease;while those which do not do so should be without effect.The early advocates of the lactic-acid theory believed that the administration of alkalies,by neutralising the acid,would cure the malady.This should have,were the theory true,been the case.But it was not,and the alkalies were found to exercise little or no control over the disease.When administered,even to saturation of the system,the patient derives no benefit therefrom;the malady lasts as long,and runs the same course,when treated on the alkali plan as when no remedies are prescribed.Were the affection produced by the acid the contrary should obtain.It has also been found that salicin and salicylic acid very quickly cure the disease,and it is obvious that they cannot do so by neutralising the lactic acid. Their effect is produced also independently of any action on the eliminating organs.It is certain that these drugs neither neutralise nor get rid of the acid,for in cases of acute rheumatism which are thus cured the perspiration often continues to give an acid reaction for four or six or more days after the fever,pain,swelling, and all the symptoms of the disease,except this acidity, have gone.This continued acidity of the perspiration so long after the rheumatic symptoms have ceased is in all probability due to the presence and excretion of acid formed during the evolution of the affection.It seems, then,that the acid in the body is neither neutralised nor destroyed by the remedy which neutralises the action of the rheumatic poison,checks the morbid processes of the disease,and cures the latter.The mere pfesence

of lactic acid in the patient's system does not occasion the symptoms experienced,which are associated with the production of the acid in excess,and not its mere presence in excess;in other words,these symptoms and the presence of lactic acid in the system would seem to be associated together as conjoint results of the morbid process in this disease.In this way the excess of lactic acid in the system is merely one of the ordimary symptoms of the affection and not the cause thereof.Regarding the assertion that the injection of lactic acid into the system of the lower animals is followed by inflammatory symptoms like those of rheumatic fever in the human disease,it may be noted first of all that Richardson injected lactic acid into the peritoneal cavityes of cats and dogs;and from the results obtained he was convinced of the truth of the lactic-acid theory,as were not a few other observers.But,when we come to consider carefully his observations,it would seem that in none of these experimented animals was there induced a morbid condition which we would call rheumatism in man. What he found,and all he found,was that in animals into whose system lactic acid had been injected,there were observed,after death,redness of a portion of the endocardium.Endocarditis,and not rheumatism,seemed to be the affection induced.But as endocarditis is frequent, and an excess of lactic acid invariable,in acute articular rheumatism,the inference was drawn that these experiments demonstrated the accuracy of the hypothesis that lactic acid is the poison which gives rise not only to endicarditis,but also to the rheumatic fever with which endicarditis is generally associated.This is certainly begging the question;for there are important points of difference between the condition noted by our author and that which occurs in connection with rheumatic fever:so important that it is justificable to regard the results of his experiments as negative andhis inferences fallacious.What he insisted was that in both induced and rheumatic endocarditis the cause of the inflammation is the lactic acid in the blood,and that the acid gives rise to the endocardial inflammation. He unhesitatingly asserts,first,that in rheumatic endocarditis the poison of the disease is produced in the pulmonary and destroyed in the systemic circulation;second, that lactic acid could not exist in the blood without giving rise to endocardial lesions;and,third,that the action of the poison which occasions the rheumatic endocarditis is directly on the free surface of the endocardium,the poison acting as a local irritant thereon. The truth or otherwise of his entire theory must stand or fall upon his first proposition.If his statement be correct,his idea of endicarditis production cannot be denied;but if,on the other hand,it can be demonstrated that lactic acid,so far from being destroyed,is actually produced in the systemic circulation,his theory is false and must be abandoned.Sugar is formed during the process of digestion,and from the digestive organs it passes into thecirculation.Part of it is absorbed by the liver,and converted into glycogen,in which form it is stored up in the liver.During fasting it again becomes transformed into sugar,and as such passes into the circulation.Like all non-nitrogenous substances,sugar is finally tranformed into carbonic acid and water. In the course of this transformation there are produced

various intermediate substances.One of these is lactic acid,the site of formation of which it is now necessary to consider.We know know that lactic,sarcolactic,acid is one of the metabolic products of muscle.In the passive state muscle has a feeble alkaline reaction;but during contraction it becomes acid.This altered reaction if due,of course,to the formation of an acid in the muscle;and du Bois-Raymond showed that the acid formed is sarcolactic acid.His observations were confirmed by those of Heidenhain,who found that up to a certain point the quantity of acid produced increased with the amount of work done by the muscle.It has been maintained by some that the acid reaction of the active muscle is partly due to the formation of phosphoric acid.Be this as it may,there can be no doubt from the observation of du Bois-Raymond that it is at least partly due to the presence of sarco-lactic acid,and that the acid is a metabolic product of muscle and is formed during its contraction.Consequently Richardson was in error when he asserted that the acid is formed in the pulmonary and destroyed in the systemic circulation.Equally untrue is his second proposition that lactic acid could not exist in the blood without giving rise to endocarditis. He tells us that it is absurb to assume that ounces of an acid of the producing series thrown off from the skin of a patient should not be derived from his blood.Lactic acis is thus thrown off from the skin in acute rheumatism.If his assertion were correct,endocarditis should be an invariable complication of that disease.But it occurs only in a minority of cases,that is to say,in about a third or so of all.Lactic acid,therefore,exists in excess in the blood,without producing cardiac complications, in about three-fourths of the individuals affected with rheumatic fever.It is also possible to refute Richardson's third proposition,that lactic acid acts as a local endocardial irritant.If lactic acid be the rheumatic poison,and if it exercises a direct irritant action on the endocardium,that membrane should give evidence of irritation in every case of acute rheumatism;but it does so in only a minority of the cases.Again,if this were its mode of production the inflammation should be diffused over the while surface of the endocardium;but it is limited to the valves,and even in these is found only on one side,in the mitral valve on its auricular surface,in the aortic valve on its convex aspect only.Rheumatic endocardial inflammation is intimately associated with the articular lesions of this disease.The poison which causes the one causes the other,and in both cases it acts in the same way.Lactic acid cannot exert such a dual action. Further,rheumatic patients almost as often show peri- as endocarditis.The two are due to the same cause and occasioned in the same way;but the propounder of the lactic-acid theory leaves pericarditis out of account altogether,and gives of the occurrence of endocarditis an explanation which cannot cover pericarditis.Thus his idea is a mistaken one,and the results of his animal experimentation cannot be advanced to his support,for the conditions in animals are not the same as in man,and administration of the acid to the latter occasions merely diaphoresis;as dogs do not perspire no sweating is observed.Furthermore,it has been shown by Reyher that the endocardial signs which Richardson attributed to the action of lactic acid are

found in all dogs,no matter how they are killed.Richardoson and his followers failed to satisfy themselves before making their experiments as to what was the normal appearance of the endocardium in the dog after death. Had they done so,they would have found it to be exactly that which they described as morbid,and as the the result of the action of lactic acid upon it.The redness which the lactic-acid theorists regarded as pathological and as the result ofthe direct action of the lactic acid,has been shown to be the normal finding in the case of dogs in whatever way dying.Richardson's third assertion is that the administration of lactic acid to man is followed by acute rheumatic symptoms.Cantani was the first to recommend the acid for the treatment of diabetes,and in not a few of the cases so managed symptoms indistinguishable from rheumatic fever,with pain, arthritis,and pyrexia,were observed.Foster reports a singular instance of this,there being six pronounced arthritic attacks.According to him,the symptoms corresponded in all respects to those which are characteristic of acute articular rheumatism,their occurrence being noted when the acid was administered and their cessation when it was abandoned.When moderate doses were tolerated,an increase in the dose was succeeded by the painful inflammation of the joints.Coincident with the arthritis was the development of perspiration,at first only slight,but afterwards,in the more severe attacks,copious and acid.Consequent of all this Foster became one of the warmest advocates of the lactic-acid theory of rheumatism.But these cases can be explained to the vanquishment of the theory named.Lactic acid is an excretory product resulting from the metabolism of the tissues.Such products when retained unduly in the system exercise two distinct action:first,a stimulant action on the organ by which they are normally eliminated,and,second,a disturbing action on the tissues which supply the materials from which they are formed. The retention,for instance,of an excess of carbonic acid in the system causes,first,increased force and frequency of respiration,and ultimately paralysis of that function and asphyxia.Excess of urea in the blood causes,first,increased action of the kidneys,and ultimately that condition of tissue asphyxia to which the name uraemia is applied.So with lactic acid,its retention in the system causes,first,increased action of the organ by which it and its products are normally eliminated, and,second,functional disturbance of the textures during whose retrograde metamorphosis it is formed,if for any reason there is a failure on the part of the emunctories.It has been found that lactic acid is eliminated chiefly by the skin,and,second,that it is formed during the action and retrograde metamorphosis of the tissues of the motor apparatus.The effects which would be expected to result from an excess of lactic acid in the blood are,therfore,increased action of the skin,and, failing that,functional motor disturbances,as observed by the author named.The acid is so readily oxidised and eliminated that it is only in rare instances that ingestion can exceed elimination.Consequently it is very seldom that its internal administration can occasion disturbance.One of these exceptional cases is that described by Foster.The patient was suffering from diabetes and pulmonary tuberculosis,both of them affections

accompanied by imperfect oxidation..Now if we give lactic acid to a patient in whose system it cannot be oxidised,and by whose skin it cannot be eliminated,it must be retained,and give rise to disturbance of the nutrition of the textures of whose metamorphosis it is a product.An excess of this acid in the blood checks the retrograde metamorphosis of these textures,and so disturbs the entire nutrition of the same,just as retained excreta produce a corresponding action on the brain,and as an atmosphere of carbonic acid interferes with the elimination of that gas from the system. The symptoms resulting from the retention of lactic acid in the blood are,therefore,likely to be those of functional disturbance of the motor apparatus.Functional disturbance declares itself in different ways in different organs.In the brain it causes nervousness,irritability, headache,giddiness,convulsions,and coma.In the heart it gives rise tise to more or less disturbance of the rhythm and force of its action.In thedigestive organs it declares itself by evidences of imperfect and deranged digestion.In fibrous tissue the evidence of its existence is pain,which may be severe,and is well exemplified by what is felt when a ligament is unduly stretched, or when it is the seat of acute inflammation as in rheumatic fever.An irritating principle like lactic acid, which acts on both the muscular and the fibrous tissues, will declare itself by symptoms referable to the latter. It will do so because those tissues give more ready and decided evidence of functional disturbance,and because any weakening of the muscles to which it might give rise would be lost in the muscular debility and wasting characteristic of the diabetes for which the exhibition of the lactic acid was initiated.Consequently pain in connection with the muscular and fibrous tissue of the motor apparatus is the chief symptom which would be expected to follow the retention in the blood of an excess of lactic acid.This is just what obtains,and is true explanation of Foster's observations,which latter demonstrate that the acid causes increased action of the skin,and,failing this,disturbed nutrition of,and consequent pain in,the muscles and in the fibrous tissues of the articulation.Despite this,one is not justified in admitting that excess of lactic acid in the blood is the cause of rheumatic fever,though it is a symptom of the disease.This is very different from allowing that it causes all the morbid phenomena,which is what the lactic theorists maintain.They say that lactic acid is the rheumatic poison,the special agent which initiates and operates all the symptoms of the disease.One of the most constant of these phenomena is the presence in the blood of an excess of lactic acid.But the latter cannot be the cause of its own formation in excess.The position is a ridiculous one.Indeed,these theorists have taken one of the symptoms of the disease and have given it the interpretation of an exciting cause.Thereby they have erred grievously.They assert that excess of the acid may occasion the articular pains,but cannot demonstrate how the excess of the acid is formed. In Foster's famous cases the acid was given to the patient and its presence in excess was readily accounted for. In rheumatic fever the excess of lactic acid is the pehnomenon which,of all others,is at once most essential and difficult to explain.If the first requiste to

the production of rheumatism be an excess of lactic acid, the first requisite to a satisfactory theory of the disease is that it should account for this excess. An attempt to do so has been made by some of the lactic-theorists, who affirm that the acid is formed during muscular exercise. Under ordinary conditions it is partly oxidised and eliminated as carbonic acid and water; partly, when there is a lot of it, excreted unaltered in the perspiration. Should the general surface of the body become chilled, the elimination of the acid will be interfered with, and systemic accumulation will occur. Itis certainly true that rheumatic fever may follow a chilling of the cutaneous surface heated by exercise. But to be heated in t is way is so common at the age at which rheumatism chiefly occurs, and to be exposed to cold so common in the climates in which it most prevails, that great allowance has to be made for the elements of chance and accidental coincidence. But making allowance for these, there can be no doubt that overheating and subsequent exposure to cold, and even such exposure without previous overheating, do in some cases seem to determine an attack of acute articular rheumatism. But it does not follow that the determining agent is the checking of the action of the skin. Overheating and subsequent chilling are not the only effects of exercise and subsequent exposure; they are not even the most common. A more constant and more important one is exhaustion, and the probable explanation of the connection which obtains between the exercise and rheumatism is that the exhaustion consequent on the exercise renders the system more susceptible to the action of the rheumatic poison. The same thing would happen from the depressing influence of cold by lowering the resisting powers of the individual. In not a few affections there has been noted a greater liability to the action of certain morbific agencies when the system is exhausted or depressed from any cause. Thus, in the case of malarial fever to which rheumatism is analogous, exhausting exercise and other debilitating influences, errors of diet, and particularly catching cold, increase the predisposition so much that persons who have long been exposed to malaria with impunity are not affected by it till one of these causes has operated. In the case of rheumatic fever it is not so much the exercise as the exhaustion which follows it, it is not so much the chilling of the surface as the depressing action of the cold on the system, which are the disturbing agencies. They do not themselves give rise to the disease, but render the poison thereof more capable of operating. In further disproof of the etiological influence of chilling it may be noted that chilling of the skin, when heated by exercise, is frequently had recourse to with impunity, if not with actual benefit. Then, again, if we regard sudden chilling of the surface as a source of danger, and as a cause of acute rheumatism, it is impossible to satisfactorily explain the beneficial action of the cold bath in the hyperpyrexia of the malignant cases of that disease. Here we have the malady presenting itself in the most distinct form, lactic acid is being freely thrown off by the skin, the patient is in imminent danger, and yet the only thing which does him good, the only thing which seems to give him a chance of recovering, is to apply cold to the surface of his body, thereby not only lowering the temperature, but checking also the action of the skin. The

same agency which is said by the lactic theorists to cause the rheumatic fever actually is capable sometimes of curing the disease. Further they affirm that during thirty minutes or so of violent exercise there is formed in the systems of those of rheumatic constitution enough lactic acid to produce an attack of rheumatic fever; second, that every time they take active exercise such persons are liable to be laid up with that disease; and, third, that they are saved from such a calamity only by the free action of their skins. This is absurd on the very face of it. If we adopt their idea we must also believe that during the said violent exertion there is produced in the system enough of the acid to not only occasion rheumatic fever, but to keep up the symptoms of that disease for weeks, and to supply at the same time the excess of acid which is being eliminated during the whole period of the continuance of the malady. The mere statement regarding what is involved in this hypothesis is enough to condemn it. For to suppose that the whole of the lactic acid which is required to occasion an attack of acute articular rheumatism of several weeks' duration, with its accompanying profuse acid perspirations, could be produced in the system in the course of thirty minutes is to presuppose the existence in the system, at the end of the period named, of a quantity of lactic acid which, granted that acid to be the cause of the disease, would produce the most acute inflammation and kill the patient from its effects in two or three days. If we admit this, all cases of acute rheumatism must be acute and the affection productive of a frightful mortality, wjich it is not. Furthermore, if the rheumatic poison is manufactured in this way, if the whole of the morbific principle exists in the system at the beginning of the attack, which would be the case if the above view were correct, we should find the symptoms of rheumatic fever developed not gradually as in the case, but all at the same time, and also very soon after the chill, and we should have the heart involved in every case. We should find, moreover, that the free action of the skin, and consequent elimination of the acid, which characterise the disease, would be followed by relief of the pain, which is not at all what actually happens in this disease. We thus see that the slow invasion of the symptoms of rheumatic fever, the protracted and varying course of the affection, the shifting character of the articular lesions, and the long-continued hyperacidity of the secretions, cannot possibly result from such an excess of lactic acid as could be produced in one or in a dozen hours of violent physical exertion. Indeed, during an attack of rheumatic fever there is eliminated by the skin alone in a single day more of the acid than exists in the system at the end of the period of exercise named. It is wrong to look on the excess of lactic acid, which certainly does exist in acute rheumatism, as simply an accumulation. The use of the term, and the undue importance attached to the operation of cold, have led to a misinterpretation of the symptoms observed. What we mean when we say that an excretory product accumulates in the system is that it is being formed but not properly eliminated. But in acute rheumatism there is increased, and very greatly increased, elimination as well as increased formation; and this increased elimination goes on during the entire evolution of the

disease.The most severe cases,those in which there is most suffering,and most of the characteristic articular trouble,are also those in which the perspiration is most profuse and most markedly acid,and in which the acid is therefore most freely eliminated.We cannot say that in such circumstances there is any accumulation of lactic acid in the system;for it is being eliminated in unusual quantity.Such increased elimination implies, not accumulation,but increased formation;and this is the phenomenon which calls for attention and explanation. Increased formation of lactic acid is one of the essentials of acute rheumatism;and no theory of that malady is satisfactory which does not recognise and account for this excess.Richardson and his followers cannot do this.His theory also both fails to explain and ignores some of theother features of the affection;for in propounding a theory of the causation of rheumatic fever it is necessary to account not only for the occurrence of the articular pains, but for the occurrence of the symptoms which precede these special and characteristic manifestations of the fully developed disease,and precede even the evidence of the existence of an excess of lactic acid.A case of rheumatic fever does not not present all its classical symptoms immediately;for they are preceded often for two or three or more days by shivering,malaise,and a sense of weakness accompanied by aching of the limbs.These initial symptoms are as much a part of the ailment as the articular pains which they usher in,and have equally to be accounted for by the said theorists.They are unable to satisfy out importunities.Nevertheless,though we ridicule the lactic-acid theory of the disease, we must admit that the acid is not without action in the production of the clinical phenomena observed.An excess in the blood of any product of retrograde tissue metamorphosis could scarcely be without some action;and there can be no reasonable doubt that the profuse perspirations which form a characteristic feature of the malady are mainly due to the stimulant action on the skin of the excess of lactic acid in the blood.It is probable,also,that should formation exceed elimination,the resulting excess of lactic acid might have the effect of exaggerating the lesions of the fibrous textures already present,and so make all the symptoms of the disease very much worse than they otherwise would be.

URIC-ACID THEORY.

Much of what has already been said applies to this theory which finds one of its strongest advocates in Haig,who attributes rheumatism in many cases to the influence of improper diet,drugs,etc.,and maintains that heredity has but little influence beyond the transmission of food habits.He finds that sodium salicylate can increase the uric-acid output by thirteen times. This is possibly due to its overproduction,for treatment by giving large quantities of water and alkalies alone does not wash out more uric acid than normal.He also finds that iron and other metals capable of forming insoluble urates are harmful in rheumatism,whereas those things which,like the alkalies,colchicum, and salicylic acid,increase the solvent power of the blood are uric acid do good,and vice versâ.In response to the denial of the presence of uric acid in the blood,which was made by some of the older writers,like Garrod,and

others also,Haig adduces the fact that modern methods of testing for the acid are far superior and more delicate;but his theory fails to explain satisfactorily why uric acid should cause tophi and certain typical visceral lesions in gout,which are wholly absent from rheumatism.Certainly,the clinical phenomena of the two diseases,rheumatism and gout,are too widely distinct to make it easy to believe in the common origin in the same morbific process.

HEPATIC THEORY.

One of the older hypotheses advanced,but which has met with little or no support,is that rheumatism and allied disorders all take origin from perverted glycogenic function of the liver.Latham and other authors have argued that the essential pathology is to be found in the LYMPH,and in a stagnant circulation in the lymph spaces,so that the nutrient fluids around the articular structures,muscles,and nerves are imperfectly renewed. Whatever be the mature of the poison,it is believed that it possesses a selective irritative action upon the fibrous tissues of the body,and that these tissues, being feeble,are irritated by it.This is most strikingly shown inthe inflammatory reaction of the various joints and the tendons and fasciae and muscular sheaths, as well as in such serous membranes as the pleura and pericardium.The irritant also disturbs the nerve centres,the fibrous,chordal,and valvular structures of the heart,producing endocarditis,and sometimes the body surface,givin g rise to erythema,nodes,and so forth.

HYPEROXIDATION THEORY.

The same objections urged under the lactic-acid theory apply to Latham's(Brit.Med.Jour.,ii,1880,p. 977)hypothesis that hyperoxidation of the muscular tissue is the starting-point of acute rheumatism.He assumes,with other physiologists,the existence of a nervous centre which inhibits the chemical changes that would take place if the tissues were out of the body.If this centre be changed or weakened,the muscle, instead of absorbing and fixing the oxygen and giving out carbonic acid,disintegrates;lactic acid is formed, and,passing into the blood,may be there oxidised and produce the pyrexia of acute rheumatism.It need hardly be remarked that the existence of a chemical inhibitory centre has yet to be proved,although much may be advanced in its favour;and,secondly,the investigations of Zuntz render it highly probable that in all febrile affections it is the muscles chiefly,if not solely, which suffer increased oxidation,and that is due to increased innervation - views not easily reconciled with the theory of the above-mentioned hypothecator.

NERVOUS THEORY.

According to the advocates of the nervous theory of acute rheumatism the mere chemical peripheral irritation of the nerves by a simple ingredient, such as lactic acid,is insufficient to directly cause all the phenomena of the disease,and there exists a a neurosis,that is,some functional nervous disorder, excited by exposure to cold or toxic substances,which, through trophic impulses,modifies the joint tissues, rendering them more susceptible to irritation and inflammation;it is the combination of theory of special nerve action with that of toxins or organic chemical irritants of the body which constitutes the neuro-

chemical or tropho-neurotic theory.It would appear that the nervous theory of rheumatism and of articular diseases originated with Dr.J.R.Mitchell of Philadelphia (Amer.Jour.Med.Sci.,1831;ibid.,1833),and was afterwards elaborated by Froriep(Die Rheumatischen Schwiele,Weimar, 1843),in 1843,Scott Alison(Lancet,1846,227) in 1846, Constatt(Spec.Path.u.Therap.,1847,p.609)in 1847,Gull in 1858,Weir Mitchell(Amer.Jour.Med.Sci.,April,1875,Vol. lxix,339-348)in 1864,Charcot in 1872,and by many others since.According to the theory,the exciting cause of rheumatism,cold,either acts directly upon the vasomotor or the trophic(?)nerves of the articulations,and excites inflammation of them,or else it irritates the peripheral end of the centripetal nerves,and through these excites actively the vasomotor and trophic nerve centres.The local lesions,on this hypothesis,are of trophic origin; the fever is due to hyperacidity of the centres supposed to control the chemical changes going on in the tissues;the excessive perspiration to stimulation of the sweat centres;and so on.It is not held that a definite centric lesion of the nervous system exists in rheumatism,analogous to the lesions which in myelitis or tabes dorsalis develop the arthropathies of those affections, but rather a functional disturbance.One of the warmest advocates of the nervous theory of rheumatism in all its forms,-simple,rheumatoid,gonorrhoeal,etc.,Jonathan Hutchinson(Trans.Internat.Med.Congr.,1881,ii,93)calls it a catarrhal neurosis,the exposure of some tract of skin or mucous membrane to cold or irritation acting as the incident excitor influence.In order that the peripheral irritation shall thus induce inflammation of the joints and the other affections of the muscles,tendons, fasciae,etc.,which are called rheumatic,he holds with the French school that the arthritic diathesis must exist,or that state of tissue-health which involves a tendency to temporary inflammation of many joints or fibrous structures at once,or to repeatedly recurrent attacks of inflammation of one joint or fibrous structure.If Hutchinson be understood correctly,he also holds that a nerve-tissue peculiarity exists which renders persons liable to rheumatism.He does not indicate either the cause or the nature of the nerve-tissue peculiarity.But modern pathology teaches that the functional conditions of the nerve centres known as neuroses, whether inherited or acquired,reveal themselves as morbid manifestations of nerve function on the part of the special portions of or the entire nervous system;and,as Duckworth has well pointed out,these neuroses may be originated,when not inherited,in various ways,as by excessive activity of the nervous system,by prolonged or habitual excesses,etc."Thus,undue mental labour,gluttony,alcoholic intemperance,debauchery,and other indulged evil propensities in the parent come to be developed into definite neurotic taint and tendency in the offspring"But some have urged thatthere is something more in acute articular rheumatism than an inflammation of certain structures,articular and visceral,lighted up in an individual of a neuro-arthritic diathesis,they looking to the closely-allied affection,gout,which involves pretty much the same structures as rheumatism, and which is held by not a few writers to belong to the same basic diathesis as it,for an suggestions. Duckworth(Brain,April,1880)rather ably advanced a neurotic theory of gout,but it was admitted on all hands,

and by Duckworth himself,that in gout a large part of the phenomena is due to perverted relations of uric acid and sodium and to the presence of uric acid in the blood.His contemporaries asked if they could not from analogy,as well as from other evidence,infer that in that so-called neurosis,rheumatism,a considerable part of the phenomena is due to perversions of the processes of assimilation and excretion,and to the presence of some unknown intermediate product of destructive metamorphosis - lactic or other acid.This was admitted by numerous authors;and in this way the pathology of the disease was made to embrace the humoral as well as the solidist doctrines - the resulting theory being a neuro-humoral one.The pure nerve origin of rheumatism was not so warmly upheld as were other theories,mainly on the ground that it fails to convincingly account for all the varied irritations present in typical cases.Its advocates point to analogous arthritides occurring in nervous diseases,such as myelitis,locomotor ataxia,syringomyelia,and sometimes with St.Vitus' dance. It is established beyond the possibility of a doubt that articular affections may occur from lesions of the nervous system.Charcot,speaking with special reference to their development in connection with cerebral disease,points out that they are chiefly found in cases of hemiplegia where the paralysis is consequent in inflammation of the brain or its softening.Brown-Séquard, referring to the pain sometimes experienced when paralysed limbs are pressed upon or moved,says that such pains depend upon a subacute inflammation of the muscles or joints which is often mistaken for a rheumatic affection.J.R.Mitchell long ago called attention to articular changes occurring in consequence of injuries of the spine.His son,Weir Mitchell,later described the curious inflammatory states of the joints which follow injuries to nerves,especially those of the arm.With the progress of knowledge it became more and more apparent that arthritis deformans was a disease by itself, quite distinct from both gout and rheumatism in both its history and symptoms;and the special attention which as it a separate ailment it received led writers more and more to adopt the view,originally advanced by Remak,that the articular changes which form its characteristic feature are in some way due to a nerve cause. The enunciation of the nervous theory of rheumatism was the inevitable outcome of the accumulation of information regarding the localisation of cerebral function and of the part played by the nerve centres in controlling and regulating the functions of organic life,in addition to the distinct evidence that serious articular lesions do take place as a consequence of lesions of the nerves and their centres,and the belief that arthritis deformans was of nervous origin. The vast majority of those who maintained that the nervous system is responsible for the production of the disease believed that the disturbance of this region which originates the articular affections is produced by peripheral irritation applied to the superficial nerves,and transmitted by them to the trophic centres of the joints.That articular lesions might thus be induced is possible;but that rheumatic fever could be so caused is highly improbable.For,in the first place,acute inflammation such as that which

characterises the articular lesions of that disease is not the form of joint disease which is associated with lesions of the nerves and nerve centres. Nor can it be regarded as a priori other than highly improbable that such acute sthenic inflammation should be the result of malnutrition. And, in the second place, inflammation of the joints is not the only thing with which we have to deal in rheumatic fever. The disease has other features and other phenomena calling for explanation as much as do the articular affections. If, for instance, as is by some maintained, cold be the common cause of the peripheral nerve irritation which causes the disease, it is puzzling to understand why the affection is more commonly observed in temperate than in cold countries; why it is most frequent at the age at which the power of resisting cold is greatest, that is to say, from fifteen to fifty; why it is so rare amongst children and older people whose power of resisting cold is so much less; why there is invasion of fresh joints after the patient has been warm in bed for days or weeks; why endo- or pericarditis should occur; why the endocarditis is so limited in extent; why it affects the valves of the left heart, and only one surface of the valve; and why the system contains more lactic acid than normal. But the nerve theorists take no heed of the cardiac affections of rheumatism, and are unable to explain some of the most striking phenomena of the malady by their peculiar argumentation. Indeed, this is not to be wondered at, for no theory which does not recognise the action of a poison, which is produced in the system during the evolution of the disease and circulates all over it, can account for the numerous and varied features of this disease. This dawning upon Latham, a warm advocate of the nerve theory, he vaunted the hypothesis that as a result of exposure to cold there is produced a hyperaemic condition of the muscles, and that from this there results an increased formation of glycogen, that this is ultimately transformed in the liver into uric acid, and that this uric acid it is by which by impairing the action of the trophic centres of the joints causes the inflammatory lesions of rheumatic fever. But it may be urged against this hypothesis that the facts which are opposed to the nervous theory also apply, and that uric acid does not exist in excess in the blood in acute articular rheumatism, which affection, of all others, seems to have least disturbance of the nervous centres. In the acute exanthemata, pneumonia, etc., there are usually symptoms which are distinctly and directly referable to disturbance of the brain. In acute rheumatism such symptoms seldom occur except in cases of hyperpyrexia and in cases complicated with acute carditis or pericarditis, and in them the nervous phenomena have nothing to do with the articular lesions, but are referable to other things. The nervous theory is also disproved by both the history and the symptoms of the disease. Before affirming that the joint lesions of rheumatic fever are trophic affections dependent on the nervous system, it is necessary to demonstrate that the actually exist as such. Gout, rheumatism, arthritis deformans, and spurious arthritis have been compared and considered by some analogous because the articulations are affected in each; but the analogies are really clinical, and therefore cannot be adduced.

MIASMA THEORY.

The miasma theory, which when it was first advanced attracted great attention, assumes that rheumatism is due to the entrance into the system from without of a miasm closely allied to, but quite distinct from, malaria. The arguments advanced were both ingenious and elaborate, though by no means favourably received in all quarters. Opposed to it are the following amongst other considerations: Heredity exercises a marked influence upon the occurrence of rheumatism; unlike malarial fever, no climate or locality is immune from rheumatic fever; the many indications that a diathesis is not inoperative in rheumatism; the remarkable influence exercised by cold and dampness in the production of the disease.

INFECTION THEORY.

Heuter was the first to suggest that acute articular rheumatism is the outcome of infection, and in this idea he was supported from the first by numerous authors and it is now pretty generally believed in In acute cases germs have been found fromtime to time in the blood-serum, synovia, pericardial fluid, and again in vegetations upon the valves of the heart, but no constant variety was uniformly found to be present, nor did inoculation experiments yield uniform results in either man or animals. Maragliano (Gaz. degli Osped. e delle Clin., June 20, 1896) later described a specific bacillus obtained from the blood of rheumatic patients with which he claimed to reproduce the disease in rabbits. The main reasons for this theory of infection consist in the following facts: First, the affection is sometimes epidemic; second, it is self-limited; third, it mainly affects the young; fourth, the severer symptoms and complications, hyperpyrexia and endocarditis and pericarditis, are suggestive of those of some other infections; fifth, there is a tendency to leucocytosis, albuminuria, and anaemia, as well as to, sixth, development of a rash, erythema, to profuse sweating and high fever, as in pyaemia; seventh, many infections, like pyaemia, gonorrhoeal or other, scarlatina, cerebrospinal meningitis, etc., present articular symptoms; eighth, toxaemia best explains the number and variety of the different symptoms. The relapses are held to be due to development of new generations of bacilli, to the action of which it is said salicylates are inimical. Duckworth, while inclined to accept this theory, holds that it fails to fully explain all the phenomena of the disease. The fact that rheumatism sometimes prevails in epidemic form is one of the strongest arguments in support of the infection theory. Mantile refers to often finding two or three cases in one household, and Newsholme has observed distinct epidemics in Norway. In such cities and places as London, Manchester, Liverpool, etc., distinct epidemics have been noted during recent years. Strümpell alludes to its periodical increase in Leipzig, and Lange of Copenhagen refers to its variations in prevalence and intensity there. Various recent writers have reported cases which appeared to be derived by direct contagion, but this is certainly not a usual experience. Sacaze claims that in many cases a prior infective wound or inflamed throat can be discovered which might afford entrance for a bacillus of rheumatism; but the majority of cases certainly present no such solution of continuity, and cases of septicaemia or pyaemia with arthritis should be distinguished from true rheumatic fever.

According to the tenets of the infection theory, the specific bacillus of rheumatism, the growth of which is favoured by a prolonged, hot, dry season, in some unknown manner enters the system of one who is predisposed by inheritance and by chilling, exposure, fatigue, debility, or the like. The entrance of the germ is attained by unknown means, but once within the system it produces toxic materials in the blood, one of which is, according to Duckworth, urice acid, or, more likely, some specific toxin, which in turn irritates the nervous system, causing various nervous symptoms and trophic disturbances, notably in the joints. A plausible working hypothesis is thereby evolved. The opponents of the infection theory claim that no germ has been found; that the disease has not been inoculated; that there is no positive proof of any case of direct transmission of infection; that there is no recognised period of incubation; yet these things were said with equal force at one time regarding malaria and tuberculosis. There has so far been no incontestable evidence forthcoming to show that acute articular rheumatism is due to a specific bacillus. True, a largish microbe was found by Achalme, in 1891, and by other continental bacteriologists subsequently, in cases of this disease and for which it was considered the cause. But these observations were not generally confirmed, which is strange in view of the fact that Achalme's bacillus was as large as that of anthrax and therefore not likely to escape detection. On the other hand, on not a few occasions a micrococcus has been accused of being the exciting cause of rheumatic fever, and with every show of reason. But here we must bear in mind that, though the affection is of a systemic nature, opinions are by no means in accord that it has only one exciting cause, numerous observers insisting that the malady is the outcome of infection with different members of the micrococcal group of germs. It has often been affirmed that rheumatic fever is a form of septicaemia. But the statement is too vague for general acceptance, as any infection, provided it be sufficiently intense, will cause blood-poisoning, and the term fails to assist us in any way regarding the real nature of the disease. There are many competent authorities who hold that the malady is caused by various pyogenic micrococci in an attenuated condition; in other words, the disease is in their opinion an attenuated pyaemia. But it may be objected to this that the affection is very common, and more so than pyaemia, and it is nonetheless very seldom seen to arise in connection with the latter, though now and then problematical cases arise in connection with middle-ear or tonsillar abscesses. But, for one of these, thousands of cases of rheumatism arise in such a way and pursue such an evolution that they cannot be regarded as instances of attenuated pyaemia. It may be further urged that rheumatic fever is not infrequently a most deadly disease, as, for instance, when hyperpyrexia occurs; and this cannot be the outcome of an attenuated infection or of a pyaemia. Further, because such such micrococci as the staphylococcus aureus have been found in the tissues and therefrom isolated in rheumatic fever, it is incorrect to say that they produce the disease; and the same remark applies to the micrococci which have experimentally occasioned articular and endocardial inflammations in the lower animals. In the case of

none of these germs have Koch's classical postulates been fulfilled.It has also been said that the cause of rheumatism is a diplococcus belonging to the group of streptococcus pyogenes,on the one hand,and to the pneumococcus lanceolatus on the other.This diplococcus has been isolated by numerous observers,such as Mantle, Klebs,Leyden,Chvostek,Singer,Popoff,Loeffler,and Michaelis.In 1898,Triboulet and Apert injected the blood of a rheumatic rabbit and produced mitral disease;and the following year Westphal,Wassermann,and Malkoff caused fever and polyarthritis in eighty rabbits with a similar germ obtained froma fatal case of rheumatic fever.Birch-Hirschfeld,Bouchard and Charrin,Sahli,and Sacaze repeatedly found the staphylococcus albus and sometimes streptococci in the synovial fluid of the articulations,in the pericardial fluid,and in the cardiac valves of cases of acute and subacute rheumatism.In numerous cases of acute articular rheumatism Leyden(Med. News,Jan.,1895)isolated a delicate diplococcus differing from all hitherto described and personally considered to be the cause of rheumatism and all its complications.Wade(Brit.Med.Jour.,April 4,1896)says that numerous cases of tonsillitis are never followed by rheumatism,while repeated attacks may never be so followed,and yet ultimately a similar attack may never be so followed;these facts,taken together,seem to strongly indicate that there is a special rheumatic bacillus or bacilli, and,further,that this special bacillus may or may not be associated with those of tonsillitis;that such an association is common cannot be denied.Singer(Berl.klin. Woch.,1897,No.31)examined ninety-two cases of acute articular rheumatism.In a large number the presence of staphylococci was ascertained.Autopsies explained why arthritic effusions in cases of acute rhrumatism are found often to be free from microbes,for in such cases the bacteria have their seat in the particular tissues only.These microbes are,according to him,probably the actual cause of acute rheumatism,which shows its pyaemic nature by its relation to erythema multiforme,sore-throat,and so forth.Jaccoud(Jour.de Méd.de Paris,April, 1897)affirms that the infectious nature of rheumatism is beyond doubt when its mode of evolution,its diffuse character,and the fact thatt there is intrauterine transmission from mother to foetus are taken into consideration.In many cases some preceding local process has been observed serving as a point of invasion to the organism,whatever it may be,which is the cause of the disease.Among these the most important is tonsillitis, and a striking fact is that the organisms found are exactly the same as those occurring in the tissues which are the seat of the location.The pharynx or tonsils - in fact,any tissues showing a lesion - may allow the organisms to enter.Riva(Cent.f.Inn.Med.,Aug.14, 1897)has endeavoured to really discover the cause of rheumatic fever.A culture medium was by him employed, the chief characteristic of which is that it contains synovial fluid taken from the joint of a horse.Employing this new culture,and using agar as a control,cultures were obtained which showed upon microscopical examination rounded bodies to which has been given the name pseudospores.According to him,these are gradually replaced by two kinds of bacilli;and the pseudospores or their bacilli are most likely the cause of acute

articular rheumatism.Thiroloix(Gaz.Hebd.,1897,No.79) has cultivated bacilli from the blood and the pleuritic exudate of a patient suffering from rheumatic fever,the culture of which prospered particularly in milk and by close exclusion from the air.The bacilli proved toxic to guinea-pigs and rabbits,provoking symptoms of a septic disease,but no affection of the joints.According to Buss(Deut.Arch.f.klin.Med.,Bd.54),the frequent coincidence of angina catarrhalis generally precedes the rheumatic affection,and both affections are caused by microbic infection.Triboulet(Rev.de Méd.,April 10,1898)says that some rheumatic manifestations are free from bacterial influence,such as thosedue to serums,cell-products, etc.No constant specific organism is found in those forms due to bacteria,and,as the result of infection by those bacteria that are active is not always the same, it is probable that in certain individuals there is a predisposition to articular diseases.Cold has an undoubted influence.This influence,however,is limited to the preparation of an already-predisposed individual for microbic invasion by lowering general or local vitality.He thinks that acute articular rheumatism is due to a bacterium with a special pathogenicity towards joints,and which rapidly loses its virulence.The staphylococcus is,he says,the most frequent agent here,both in cases of frank rheumatism and in the articular affections associated with scarlet fever,puerperal sepsis, etc.Pseudorheumatic affection he considers due to haemic infection,from some local infection,such as gonorrhoea,with articular manifestations.Chronic rheumatism he believes to be the result,when the acute attack has largely subsided,leaving only a less active process;or when the pseudorheumatic process has been prolonged, causing permanent articular changes;or when the resistance of the individual has been so great or the virulence of the microorganisms so slight that general manifestations were absent.Chauffard and Ramon(Rev.de Méd., May,1898)have examined many cases of relapsing rheumatism for glandular enlargement in cases in which an infectious origin was suspected.In not a few of the cases investigated it was found that the glands sometimes in the immediate neighbourhood of the affected joints became swelled at the time of the attack.In most cases there was some pain in the swollen glands.Cultures made on various media from one of the cases was negative, but cover-glass preparationsfrom the liquid in the joint and neighbouring lymphatic glands both showed a diplobacillus.Histologically the enlarged glands showed the lesions of lymphatics,with increase in the trabeculae of the glands.This glandular enlargement they regarded as further proof of the infectious character of rheumatism.Reinhard(Münch.med.Woch.,Sept.13,1898)contends that rheumatism is an infectious disease secondary to some injury to the mucous membranes,particularly those of the mouth,which permit the entrance of the infectious agent.Rabl(ibid.,Sept.13,1898)ridicules the idea that the poison of this disease enters the system through solution of continuity in the mucous membranes.Rheumatic pains,he says,are due,with the fever,to interference with the secretory activity of the skin.According to Marshall(New York Med.Jour.,Aug.12,1899),the presence of lactic acid in the system is a predisposing cause of rheumatism,though there is no conclusive evidence

that it is the exciting cause;the disease is characterised by reduced alkalinity of the blood,and caused by toxic agents whose character and identity are yet a mystery.In 1891,Achalme(Brooklyn Med.Jour.,June,1900) found an obligate anaerobic bacterium in a state of purity at the autopsy of a man who had died of cerebral rheumatism.This organism,a large rod resembling the bacillus anthracis,staining with the aniline dyes and not decloroising under Gram's method,has been found by other observers nine times.Sodium salicylate added to the medium hindered the development of the cultures.The medium in which the bacillus grew tended rapidly to become acid and unable to preserve the vitality of the organisms.Alkalinisation of the medium by calcium carbonate prolonged their activity.Poynton and Paine(Lancet, Sept.29,1900)found a diplococcus in eight successive cases of acute rheumatism,and in five cases in pure culture.The organisms were obtained from the blood of living patients suffering from acute rheumatic pericarditis,from the pericardial fluid and from the fragments of granulations removed from the valves after death,and also from the throat of the living patient suffering from rheumatic tonsillitis.They were also isolated and grown in an acid medium,and also upon blood-agar;they were grown,too,in the pericardial fluid,which on these occasions proved to be acid.They did not thrive on ordinary media and were isolated in pure culture from the articular exudation,cardiac blood,urine from the bladder,and cerebraspinal liquid of rabbits that had been inoculated with a sufficient dose.The organism seemed to be identical with that described previously both by Triboulet and Wassermann.The microbe in question is a small micrococcus, 0.5μ in diameter,and generally grows in couples or in short chains.A capsule is not usually observed,though in the case of human beings Poynton thinks that such exists.It tinctorially reacts to all the aniline dyes.Certain observers hold that it retains Gram's stain;if it does so,tenacity of purpose is not marked.The organism becomes swollen or pear-shaped in its degenerative forms.Paine called it the diplococcus rheumaticus,the term streptococcus being inapt for descriptive purposes,though suitable to designate the family to which it belongs.Poynton says that in bouillon at 37.C.there is in twenty-four hours turbidity with slight flocculent deposit;the latter becomes distinct in three days when the fluid clears;acidity of the medium is observed.In gelatine stab cultures within forty-eight hours minute colonies develop along the track of the needle,but there is no liquefaction. The most suitable medium is blood-agar,which is prepared by smearing fresh blood upon agar and incubating for twenty-four hours.In twenty-four hours after inoculation tiny white colonies make their appearance,which remain separate from one another and turn the haemoglobin to a rusty-brown colour.Upon this medium the vitality of the organism is very striking,and the growth may be prolonged for years in subculture.A medium composed of equal parts of milk and bouillon,slightly acidified with lactic acid,is very useful for isolation purposes,the milk being coagulated in twenty-four hours. Blue litmus milk is turned pink in twenty-four hours, and the milk itself is coagulated.Vernon-Shaw considers that the best medium is glycerine,,veal broth containing

two per cent.peptone,and one per cent.alkaline to phenolphthalein.Beattie(Brit.Med.Jour.,Dec.22,1906)says that he found a definite and very distinct reaction in the production of acid and the precipitation of the bile salts in McConkey's bile salt lactose broth.The capability of producing acid is one of principal characteristics of this microorganism;it does so to a greater degree perhaps than any other germ.The acids produced vary according to the medium in which it is grown. According to Walker and Ryffel,a large amount of formic acid is produced,and this they managed to extract from the bodies of the microbes themselves;this is very suggestive in view of the well-known irritant effects of formic acid upon the textures of the body.Poynton thinks that,despite the difficulty sometimes encountered in demonstrating them,they are probably present in all the principal rheumatic lesions,and says that they have been demonstrated in the endocardial and pericardial tissues,urine,blood,pleura,peritoneum,pia mater, synovial membranes,nodules,and lungs.He refrains from calling the organism which he observed specific of this disease,though he is of the opinion that it may be so regarded.Newsholme(Practitioner,Jan.,1901)adduces certain arguments as to the infectivity of acute articular rheumatism.The clinical features of the disease and its analogy with recognised specific febrile affections confirm the view that it belongs to the same group. The mode of onset,the frequent occurrence of preliminary sore-throat,and the course of the fever point in this direction.It shares its tendency to relapse with such maladies as diphtheria,scarlatina,typhoid fever, and influenza.The liability of second and later attacks does not preclude this conception of the disease.There is,among diseases admittedly infective,a regular scale of immunity following a first attack from smallpox,in which it is nearly absolute,though enteric fever and scarlatina,in which it is feebler,to diphtheria in which immunity is evanescent,and down to erysipelas,in which one appears to predispose to further attacks.Rheumatic fever comes at this end of the scale.Nor can it be said that family inheritance argues against the infective character of rheumatic fever.The special proclivities of certain families to diphtheria,enteric fever, and scarlet fever is notorious.That a special proclivity is required to develop the introduced virus of rheumatic fever may be admitted,but this does not preclude its infective character any more than in the analogous case of erysipelas.The apparent absence of infection from patient to patient is explicable on the ground that the contagion is buried in the infected joints.Direct personal infection is relatively rare in typhoid fever and cholera,in which diseases the contagium has exit from the patient.It is likely that the majority of microbes causing rheumatic fever pay for their hardihood in invading the system by becoming destroyed within its cells.The fact that the joints are the common seat of the trouble favours the infective theory.As Payne puts it,the"vessels of the joints appear to have some special proclivity to form a nidus for the wandering germs of disease!"The therapeutics of the affection confirm the same view.The specific power of salicin in rheumatic fever is comparable to that of quinine in malaria and of mercury in syphilis.St.Clair Thomson

(ibid.,Jan.,1901)sums up the present state of our knowledge on the relation of tonsillar affections to rheumatism as follows:First,it is certain that some cases of acute rheumatism are preceded by an angina in a proportion varying from thirty to eighty per cent.Second,both rheumatism and angina have many etiological points in common,such as season of the year,cold,wet,fatigue,depressing influences,vitiated air,etc.Third,The connection of angina and rheumatism,though undoubted in a number of cases,is not yet clearly established.Fourth,the tonsil may be the port of entry of the rheumatic poison, and this even though the naked-eye appearance of the throat gives no indication of its being affected.Fifth, The particular affection of the throat which is associated with rheumatism is not yet established.Apparently it is not peritonsillar abscess.Sixth,peritonsillar inflammation does not appear to be arrested by the administration of antirheumatic remedies.Many cases of parenchymatous and lacunar tonsillitis,on the contrary,are considerably benefited by the administration of salicin or salicylate of soda.That this action proved the rheumatic nature of the disease cannot yet be accepted.Seventh,the question requires further research in two directions:One in differentiating the various forms of angina and determining the one which is associated with rheumatism;the other in further research to discover the true nature of rheumatism.F.Meyer(Deut.med.Woch.,Feb.7, 1901)has studied the mucus from the tonsils in cases of rheumatism.He found diplococci in rheumatic cases,but not in others;they produced a seropurulent,usually sterile,exudate in the joints,which did not proceed to sepsis.These bacteria have a peculiar affinity to the serous membranes,and the endocardium in particular.This makes it probable that they have a close relation to actual articular rheumatism.Menzer(ibid.,Feb.14,1901) informs us that the discovery of streptococci in rheumatism is not new,though this does not mean that it is without importance.Streptococci and staphylococci are frequently found in almost any infectious disease,and particularly in the mouth and throat.The important feature of some of these organisms,however,is a tendency to cause disorders in the joints.Whether streptococci can be considered as the sole cause of rheumatism or whether the streptococci at times found in normal tonsils lack this tendency to produce articular changes,it is impossible to say.Mann(Medicine,July,1901)thinks that the true cause of rheumatic fever is an infective microbe,most probably a diplococcus;that it has an affinity for serous membranes,and that this same diplococcus is the etiological factor in malignant rheumatic endocarditis,often in endocarditis valvularum,probably of chorea rheumatica,possibly in certain forms of pleuritis,and probably in some forms of peritonitis.This organism may not be the sole cause of acute polyarthritis rheumatica.That there may be others is to be inferred from the etiologcal relations of the gonococcus.Lartigau (Albany Med.Annals,May,1902)considers that rheumatic fever is an infectious disease very probably produced by a specific nacterial excitant.He insists that the claims of Achalme and others that the infection is attributable to an anaërobic bacillus have not been substantiated and are very probably untenable.The correctness of the contention advanced by Singer that the

disease is a modifed pyaemia is very doubtful.It is probably much safer to say that secondary infection with pyococcal bacteria is common in this disease.The diplococcus isolated by Wassermann,Poynton,and Payne,and several others,is probably a modified streptococcus.All of the inoculation results induced by this assumed diplococcus may be obtained with the streptococcus pyogenes. The demonstration of this organism,then,as the causative factor of acute articular rheumatism is incomplete. The specific bacterial excitant of the disease still remains undiscovered.Haig(Practitioner,Feb.to April, 1891)concludes regarding the influence of urice acid as an active and efficient influence in the production of acute articular rheumatism as follows:First,that any diminution of the alkalinity of the blood and tissue fluids in a given region of the body causes the urice acid coming to it in the blood to become less soluble and more easily retained;in other words,causes it to remain in the fluids of the less alkaline region,instead of passing on in the circulating blood.The blood thus becomes poorer,and the region of diminished alkalinity richer in uric acid or biurate.Second,according to Garod,certain regions and tissues,as the liver,spleen,and the cartilages and fibrous tissues of the joints,are normally less alkaline than the other tissues of the body and their fluids;hence,in any general diminution of alkalinity these tissues will be most affected, and the circulating urice acid will first of all be rendered insoluble or retained in them.Third,it follows from what has been said that the uric acid,or biurate, thus concentrated or precipitated in certain tissues, gives rise to irritation,going on to inflammation,which is roughly proportional to the amount of urice acid concentrated in any given spot,and the time during which it can act upon the tissues.It may be noted that when any tissue or organ is thus collecting or retaining,so to speak,all the uric acid that comes to it in the bloodstream,and while the local pains in the tissues concerned are increasing,the blood grows poorer in urice acid; as a normal consequence of this,the amount excreted in the urine diminishes,so that we have an independent source of evidence as to what is going on.Conversely, when an alkali or other solvent of uric acid has been introduced into the circulation,the process is reversed;the blood now passing through the irritated tissue now takes up in solution the uric acid that was previously retained or deposited;the affected organs or tissues grows poorer in uric acid,while the blood grows richer;and,as an evidence of this latter change,there is an increased excretion of uric acid in the urine. By assuming that the chief predisposing causes of rheumatism diminish the alkalinity of the blood,or of the fluids of the local tossues,it is plausibly explained in the foregoing conclusions how the uric acid,rendered less soluble,is attracted to and made to accumulate in the tissues sufficient to cause pains rheumatic inflammation,while the blood in general circulation and the urine would yield less than natural,when subjected to the usual tests.Porter(Amer.Med.-Surg.Bull., Jan.,1893)says that lactic acid is the cause of the active symptoms of rheumatism.An excess of this acid is generally produced by eating too freely of food containing a large percentage of starch and sugar or

proteids,and thereby introducing more than can be completely oxidated.By such incomplete oxidation of the proteid compounds within the system,the percentage of urea within the system falls,uric acid increases,and lactic acid appears in large quantities;and to this might yet be added a long list of other by-products. But it is the lactic acid which chiefly appears in rheumatic affections.Mordhorst(Cent.f.Inn.Med.,Nov.19, 1898)from chemical experiments concludes that the phenomena of rheumatism depend upon the accumulation of urate spherules in the connective tissue and cartilage. The alkalescence of juices of connective tissue is due to sodium carbonate,not the bicarbonate or phosphate. Acids favour,in the extreme,the precipitation of the spherules,while alkalies are in the opposite extreme of preventing this precipitation.Sodium salicylate favours the transformation of urate spherules into urate needles.This explains why this salt cuts short the inflammatory process,but predisposes to relapse,which is due to the needles' persistence in the tissues.Urate spherules are naturally subject to oxidation;urate needles are not.Sodium carbonate and bicarbonate do not directly hasten solution of needles,but probably favour oxidation of spherules.These salts,he says,are best given in mineral waters free from lime and containing sodium chloride and carbonic acid.

MORBID ANATOMY.

JOINTS.

The pathological anatomy of acute articular rheumatism is in no wise distinctive.Most patients recover perhaps with permanent lesions in the joints, and,unless,cardiac valvular disease follows as a sequel to endocarditis,they are wholly free from pathological remains.It is generally admitted that the process is an inflammation involving chiefly the synovial membrane,and to a less degree the cartilages,ligaments, tendinous sheaths,and in some cases even the bones and periarticular soft parts.The synovial membrane is more or less injected and reddened diffusely or in patches,especially where it forms fringe-like folds and at its line of union with the cartilage.It is somewhat thickened,opaque,and devoid of its satin-like lustree, and in somewhat protracted cases covered here and there with a thin,easily detached fibro-membranous formation. Within the articulations will be found a few drops to one or two ounces of a fluid,pale,citron- or reddish coloured fluid,like synovia,but more fluid,and generally turbid and containing transparent or semi-opaque gelatinous masses or albumino-fibrous flocculi. The microscope reveals in the effusion large detached spherical epithelial cells in various stages of germination or of fatty degeneration,and a variable number of red blood-corpuscles and pus-cells.Very exceptionally,

the effusion is mixed with more or less true pus. In two out of the eight fatal cases reported by Fuller, in which the joints were examined, pus in moderate quantity was found along with other products in some, but not in all, of the inflamed articulations, and one of them was complicated with erysipelas, the other with sloughs over both trochanters. In very severe forms complicated with haemorrhagic tendencies the inflammatory products have contained a large proportion of blood. Cornil and Ranvier (Manual of Pathological Histology, Paris, 1869, 406) insist that even in slight cases of rheumatic arthritis the diarthrodial cartilage constantly suffers changes arising from nutritive irritation and proliferation of the cartilage cells. At first the cartilage loses here and there some of its polished hyaline appearance, and the microscope reveals a finely-striated condition of its structure which gives it a velvety aspect. When the inflammation has been more severe and of longer duration, so that the deeper layers have been involved, the unaided eye will perceive local swellings in which the natural elasticity and resistance of the cartilage are impaired, and its surface is fissured or villous-like in appearance. In certain rare cases of mono-articular acute arthritis true ulcerations of the cartilage are seen. The soft parts in the immediate vicinity of the inflamed joints may be in some cases more or less congested and oedematous, and the tendinous sheaths, and even the bursae mucosae, inflamed and distended with inflammatory products like those in the articulations. Charcot (Clinical Lectures on Acute and Chronic Diseases, Syd. Soc., 1881, p. 148), holding the opinion that arthritis deformans is but a chronic variety of articular rheumatism, quotes Gurlt's statement that in acute articular rheumatism "the medullary tissue of the ends of the bones undergoes a great increase of vascularity, with proliferation of its corpuscles", and remarks that Hasse and Kussmaul have also referred to lesions of the bone and periosteum in that disease. But the condition of the osseous parts of the joints in acute articular rheumatism still needs further investigation. In subacute rheumatism these alterations in the synovial membrane, and especially in the cartilages, are likely to be more marked than in the acute variety.

BLOOD.

The blood is very commonly affected in rheumatic fever, and anaemia is of strikingly rapid in its appearance; few diseases, other than perhaps diphtheria, can impoverish it so soon. The red blood-corpuscles are diminished by one half or more, the haemoglobin falls below fifty per cent., and there is a decided tendency to leucocytosis. The salicylates, so often given in treatment of the disease, greatly increase this anaemic condition if their use be long continued. An excess of fibrin is frequent in the blood, which may reach double the normal percentage; Devaine (Jour. de Méd. Interne, Feb., 1898, Vol. ii, No. 3, p. 75) found the percentage to rise from three to seven or eight parts per thousand. In bad cases capillary dilatation, ecchymoses, and extravasations may occur on mucous or serous surfaces, or in the skin producing purpura haemorrhagica.

HEART.

This organ is the seat of inflammation in about a third of the cases of rheumatic fever; for,

although bruits are heard in a larger percentage, some of them are due to previous attacks of rheumatism or some other condition, such as anaemia. The left side is oftenest affected, probably on account of its greater functional vigour and activity; for the poison of acute rheumatism seems to irritate the tissues while they are most vigorous, selecting the joints of younger persons by preference. The structural alterations of endocarditis in this disease consist in the proliferation of the endocardial connective tissue elements and round-cell infiltration in the subendothelial tissue. On the surface of the inflamed part fibrin is deposited, the superficial elements having undergone necrosis. These fibrinous deposits give rise to minute vegetations which rarely exceed 4 mm. in size. They are regarded as the characteristic lesion of this form of endocarditis, but they are nothing more than granulation tissue covered with fibrin. Microbes may be found at the site of these deposits. These vegetations may increase in size and become the seat of ulceration, or they may be absorbed and the endocardium become thickened. In rare instances they become detached, giving rise to emboli which cause infarctions in various organs. In the majority of instances permanent lesions of the valves result from fibroid changes. It is this tendency to fibrosis which gives to simple acute endocarditis its greatest danger. The mitral valve is more often the seat of these changes than the aortic. The changes in the mitral valve are for the most part on its auricular surface, while the changes in the aortic valve are on the ventricular surface; in other words, these vegetations are found chiefly on the surface of the valve which is opposed to the current of the circulation. They are usually arranged on the borders of the aortic valves a little distance from their edges. In the centre of the band of tissue which passes from the attachment of the valve to the Arantian body the granulations are most numerous. Upon the auricular surface of the mitral valve near the insertion of the tendons the irregular wreaths of vegetations enclose the attachment of the chordae tendineae. The tendons themselves may become so soft and fraible as to rupture, or they may become adherent to one another. When such adhesions occur, either with agglutination of the flaps to each other or to the heart walls, mitral incompetency or stenosis may result. It should be borne in mind that the chief danger in all cases of simple acute endocarditis is from the fibrotic changes which occur in the valves and their orifices.

Pericarditis commences, as in the case of other serous membranes, by an injection of the blood-vessels and temporary dryness of the membrane. The capillaries become distended and the blood-stream at first stagnates. With increase in the velocity of the blood and in the calibre of the vessels the membrane assumes the reddened appearance characteristic of inflammatory action. The capilaries rupture in places, with the production of ecchymotic spots. At the same time, the liquid part of the blood escapes and the membrane becomes infiltrated and swollen. The nutrition of the endothelium being interfered with, some of the cells die and are desquamated, others proliferate. In consequence of the changes in the vessels and endothelium the membrane loses its pearly lustre. Transudation of the liquid part of the blood continues. Meeting on the free surface of the

visceral or parietal pericardium with wandering cells, some of which have disintegrated, coagulation of the fibrin factors takes place. This results in the formation of a thin continuous layer of greater or less extent with irregular thickenings, which present the appearance of little drops of opaque liquid. These enlarge and coalesce, with a consequent increase in the thickness of the membrane, which looks, as Laennec said, like two layers of butter which have been pressed together and separated. The process may be limited to one ormore points of the pericardial surface, visceral or parietal, or be generally diffused. When the inflammation is partial in extent, its seat of election is at the base of the heart near the origin of the great vessels. False membranes are more frequently formed on the visceral layer. Inflammation of the pericardium covering the vessels may extend intotheir walls, and if limited here should be regarded as an inflammation of the aorta. This condition not infrequently is the starting-point for thoracic aneurisms. The outpouring of lymph continues, and further coagulation may lead to the formation of successive superimposed layers of fibrin, with an additional increase in the thickness of the membrane. The coagulation is generally irregular in extent, and the membrane assumes various aspects. It may be honeycombed or felted or have villous projections. The projections, however, are due to the action of the heart pressing upon and rolling out smaller irregularities of surface. The fibrinous exudate may be relatively dry, and justify the name, plastic pericarditis, but it never occurs without a liquid effusion, however small in amount. A purely serous effusion rarely occurs with pericardial inflammation. The quantity of liquid effused varies greatly. There may be only a few ounces, or it may accumulate to the extent of several pints. The colour of the liquid may be light-yellow, greenish, brown, or haemorrhagic. The presence of blood may result from an actual haemorrhage into the pericardial cavity through rupture of a small vessel. Histologically the effusion is found to contain full-sized desquamated endothelial cells, or smaller or more rounded ones from proliferation; pus cells in variable numbers, in ordinary cases not sufficiently numerous to give rise to any opacity; shreds of fibrin which have been torn off by the movements of the heart; and molecular matter which probably comes from the disintegration of pus-cells and fibrin filaments. A moderate amount of red blood-corpuscles is invariably present, but, as a rule, they do not impart any colour to the liquid. The liquid effusion is absorbed again in the event of recovery taking place. The pus and other cells, except in purulent cases, undergo disintegration and pass off through the lymphatics. A large amount of fibrinous exudation may disappear in the same manner. But pericardial inflammation generally leaves traces behind it upon the visceral or parietal layers of the pericardium. It is generally agreed that the milk patches so frequently observed at the autopsy are the result of localised inflammations which may or may not have been recognised during the lifetime of the patient. Organisation of a part or the whole of the false membrane is of constant occurrence in pericarditis. In the former case patches of newly formed connective tissue will be found on the surface of the heart, over which the endothelium may

have grown;there may be thickening of the parietal layer of the pericardium without adhesion,or there may be circumscribed adhesions between the two layers.Again, threads or bands of newly formed tissue may pass from the visceral to the parietal layer.In some instances, where the inflammation has been general,organisation extends until the pericardial cavity becomes entirely obliterated.In purulent pericarditis the two layers of the pericardium are thickened and have the appearance of a granulating surface.The pus accumulates in quantities varying from a few ounces to several pints,and is seldom absorbed.It may be discharged externally through a fistulous opening,or may break into the mediastinum or one of the pleural cavities.Occasionally the liquid part of the pus is absorbed and caseation results.The chessy matter may form the seat of calcareous deposit.In rare instances pericarditis is complicated with inflammation of the mediastinal connective tissue, and then adhesions of the pericardium externally often result.In consequence of changes on the surface of the heart,its muscular fibres undergo a greater or less amount of parenchymatous degeneration,or an interstitial myocarditis may be excited.Myocardial changes most often occur in haemorrhagic and purulent pericarditis. If the pressure upon or constriction of the coronary arteries has been sufficiently great,the cardiac muscle will suffer more generally,and dilatation may result. Some authors state that the heart is always in a state of debility after inflammation of the pericardium;after extensive adhesions between its two layers dilatation with subsequent hypertrophy is commonly observed.

Myocarditis is another complication of rheumatic fever,and according to the extent of the inflammatory process a diffuse and a circumscribed myocardial inflammation are distinguished.If the muscular fibres are mainly affected,the condition resulting is a parenchymatous inflammation,while if the inflammatory process occurs in the interstial tissue,it is called interstitial myocarditis;transitional forms are common.In acute parenchynatous myocarditis the changes called by Virchow cloudy swelling are found in the cardiac muscle.The latter appears somewhat swollen,has a dull lustre or bacon-like appearance,and is of tender,brittle consistency.On microscopical examination the individual fibres are seen to be swollen.They have lost their transverse striation,and their interior is filled with numerous fine closely packed granules.If acetic acid or dilute caustic potash is added to the misroscopic preparation, the granules disappear,and become a permanent transparent mass,thereby showing their albuminous nature.At the same time there is seen to be an increase of the nuclei of the muscle.If the disease is quite marked and has lasted for some time,the process does not remain in the stage of cloudy swelling,but the granules change to fat drops,which no longer dissolve on addition of acetic acid or caustic potash,but assume a gray or black colour with osmic acid.The muscle nuclei participate in the fatty degeneration,and of the latter is marked the muscular fibres appear as cylinders thickly filled with large fat drops,and nuclei can no longer be shown in them by means of tinctorial reagents.Acute diffuse parenchymatous myocarditis is most frequently encountered in cases where the infection is severe.

The intensity and duration of the infective process, together with the individual resistance, determine which cases are to be exempt from myocarditis. In the latter the function of the heart is necessarily impaired, and it may even terminate in cardiac paralysis, which is always an occurrence to be greatly feared. Acute diffuse interstitial myocarditis is sometimes observed, a large aggregation of round cells occurring in the intermuscular connective tissue, which in places gives rise to disappearance of the muscle fibres. Acute diffuse interstitial myocarditis is generally purulent, and probably in most cases depends upon embolism of the coronary artery or its branches. If the embolic cardiac abscesses depend on ulcerative endocarditis they are multiple and sometimes in great numbers. Their appearance changes with their age. The newest ones represent fine gray or grayish-yellow points or linesmwhich under the microscope show little else than an embolus inside a vessel, and on staining with aniline colours they are seen to be filled with low organisms. In older foci they are surrounded by a haemorrhagic zone, and in still older ones suppuration has begun in the neighbourhood. The greater the abscess the less is the number of low organisms. The size of the cardiac abscesses varies from that of a pinhead to a pea or even a dove's egg. Sometimes several abscesses lie together, or they are connected, and on section form irregular cavities. Resorption of small abscesses may perhaps be possible, leaving cicatrices behind. In larger abscesses absorption is not to be expected. In some cases the pus thickens and becomes changed to a mortar-like mass, sometimes with deposit of lime salts. Usually a capsule of connective tissue is formed round the pus, and it becomes encysted. In other cases the pus perforates, either into the pericardium, producing diffuse pericarditis followed by death, or into the heart, causing small emboli in the spleen, kidneys, occasionally the brain and skin if the perforation has been into the left ventricle. The emboli are seated in the lung if the right ventricle has been perforated. After perforation into the heart, blood is forced in to the abscess cavity, and washes it out, and distends the layers of muscle, forming an acute cardiac aneurism. The latter may rupture into the pericardium and produce death from internal haemorrhage and paralysis of the heart. Cure of cardiac aneurism does not seem to occur, probably because the process of healing is prevented by the ever-flowing blood. Sometimes the formation of a long fistulous suppurating tract precedes the perforation of a cardiac abscess, so that the point of rupture is quite distant from the location of the abscess. Thus it may happen that the pus forces its way between the two endocardial layers of a valve to its tip, and then gain access into the ventricular cavity of the heart. Chronic inflammation of the myocardium is sometimes seen in rheumatic cases, the so-called rheumatic-traumatic myocarditis. During the acute stage of the disease it may be seen, and chronic muscular rheumatism is said by some to predispose to chronic myocarditis. Sometimes the myocardium suffers a chronic change from the beginning; sometimes acute changes have preceded, which become chronic; lastly, chronic myocarditis may depend upon a preceding peri- or endocarditis occurring in the course of rheumatic fever. It seems to be more common in men

than in women,and it is seen most often in patients after forty years of age.Chronic myocarditis is characterised by its tendency to the formation of connective tissue plates or callosities:whence the name myocarditis interstitialis sclerosa.In examining the heart after death in such a case the section of the cardiac muscle should be made parallel to the course of the fibres in horizontal lines,as the extent of the change can only be made evident in this way.Cardiac callosities appear at first as grayish-red,later gray-white spots, lines or plates which pervade the heart muscle more or less abundantly.Sometimes they appeara as pale-yellow or brownish spots,which in microscopic examination consist of the remains of contracted and fatty muscular fibres and colouring matter.The extent of these callosities is very various.Sometimes they appear to be simply an increase of the intermuscular connective tissue, while in other other cases form branched and extensive plates three or four centimetres broad.Again,they are dense solid nodules.Sometimes they extend through the whole thickness of the heart muscle,so that pericardium and endocardium are separated by connective tissue only.They may be so numerous that the greater part of the cardiac muscle is replaced by them.They are found most frequently in the wall of the left ventricle, especially near the apex,and next in frequency in the septum of the ventricles.On the right side of the heart they occur for the most part during foetal life,and then often give rise to congenital heart disease.If the cardiac muscle contains connective tissue callosities of small number and extent,they may be without importance,and only be found at autopsy.If,on the other hand, they be in large number and extensive,they give rise to insufficiency of cardiac power,manifested by cardiac weakness and evidences of passive congestion,just as in uncompensated valvular lesions.In addition to the appearances of chronic myocarditis there are usually other changes in the heart.The epicardium is often cloudy and thickened,and the same thing may be found on the endocardium.Occasionally the heart muscle is hypertrophied in places unaffected by the disease.Sometimes the myocarditis depends upon disease of the coronary arteries,the intima being thickened and narrowed in places.Sometimes the process is a thrombotic closure of a branch leading to a white or haemorrhagic infarction of the muscle,necrotic softening,absorption, and formation of cicatrices.Sometimes the papillary muscles are the seat of the process.They shrink and change at their apices to stiff tendinous bands.Their function is thereby impaired,and in connection with these changes there may be shrinking of the valves leading to insufficiency.As a result of chronic myocarditis the condition known as true cardiac stenosis may occur. The connective tissue forms a ring to the point where the pulmonary artery is given off,and contracts so as to occasion considerable stenosis at the beginning of the pulmonary artery.The occurrence of this at the conus arteriosus produces the appearances of stenosis of the aortic orifice.Chronic cardiac aneurism may result from chronic myocarditis.Since the cicatrices are not capable of contraction,they gradually yield to the blood-pressure inside the heart,and form a sac with perhaps a narrow,shrunken entrance.These saccular dil-atations,known as chronic aneurisms,are most often

found in the left ventricle near the heart's apex.Sometimes there are several aneurisms in one heart,which usually lie close together.The size of the aneurisms may equal that of the heart,or even surpass it.If the cardiac aneurism has reached a certain size,all traces of muscular tissue disappear from its walls,and there may be calcification in places.Thrombi may be formed in concentric layers in it.Very frequently there are adhesions between the outer layers of the aneurism and the parietal layer of the pericardium,so that the aneurism projects into the left pleural sac or is largely surrounded by the laft lung.If the aneurisms are in the septum ventriculorum,they extend,almost without exception into the right side of the heart on account of the greater blood-tension in the left heart.

Lees(Brit.Med.Jour.,Jan.5,1901)says that in acute articular rheumatism,even in the most subacute attacks, acute dilatation of the heart seems to be invariably present.Since he first observed its occurrence,seven years ago,he has never seen a first attack of this disease,whether in a child or in an adult,in which it was absent.When the rheumatic attack is over,the dilatation lessens and the cardiac dulness may again become of normal extent.He has no hesitation in saying that acute dilatation of the heart is much more common in rheumatism,even in slight attacks,than in either diphtheria or influenza.Yet,though more common,it is far less dangerous.An extension of the cardiac dulness to two finger-breadths outside the left nipple-line is a grave danger ina child affected with diphtheria;but the same amount of increased dulness in a child suffering from rheumatism implies,in itself,no immediate danger of fatality whatever.The dilatation of rheumatism is so much less dangerous,he says,than that of diphtheria or of influenza,in spite of its considerable amount.The difference must be produced by a different effect of the several toxins upon the cardiac muscle.In diphtheria,and apparently in influenza,the muscular fibres of the left ventricle suffer greater destruction;in rheumatism the myocardial changes are less intense,and one can only suppose that the elasticity of the ventricle is more affected.Poynton's section shows that,though in the rheumatic heart there is evidence of fatty degeneration of the cardiac muscular fibres,with interstitial foci of small cells and vascular dilatation,yet the destruction of the muscle is much less pronounced than in the diphtherial heart.But though an increase of cardiac dulness to the above-mentioned extent outside the nipple-line in a case of rheumatism involves no danger of sudden death,yet a further extension,occurring rapidly, may cause decided symptoms of collapse.The slightest suspicion of rheumatism in a child should therefore lead to a careful and repeated examination of the heart.Even in adults,he says,much oftener than is generally recognised,it is fresh rheumatism that kills, breaking down compensation.It is important to notice that at the necropsy of patients who have died from chronic rheumatic heart disease,there is usually evidence of fresh endocarditis on the cardiac valves;and clinically it may often be observed that,when a case of mitral stenosis breaks down,there is some evidence of fresh rheumatism forthcoming.Regaring the pathological anatomy of the condition,we find the heart enlarged.

As a rule,however,the enlargement is not uniform,one cavity or one side of the heart being dilated. The affected cavity is not infrequently filled with dark, imperfectly coagulated blood,and its wall is thinned. The right heart dilates acutely more frequently than the left in previously healthy individuals,for the reasons that the obstruction to be overcome arises more frequently in the pulmonary circuit,and that the right heart is less liable to meet the demands for increased work because of the relative thinness of its wall.If the obstruction has occurred in the systemic vessels, the left ventricle will be found dilated,and in addition to dilatation of that chamber there may be dilatation of the auricle of the same side.

Coming now to the subject of chronic valvular cardiac disease it would appear that in rheumatic cases aortic stenosis is occasionally observed.Rheumatic endocarditis is the most frequent cause of that form of stenosis which is due to valvular changes which not only cause obstruction at the aortic orifice,but allow of more or less regurgitation.Such narrowing may be the result of any of the valvular changes which take place in the course of acute or chronic endocarditis.Fibrotic changes in the aortic cusps may render them so rigid that they cannot be pressed back against the walls of the aorta,and their underlying prominence obstructs the outgoing current of blood.If they become the seat of irregular warty excrescences,as often happens,they still more seriously interfere with the current of blood. In rare instances the aortic orifice may be diminished by fibrotic constriction at the base of the valves.Adhesions of the segments of the latter usually begin at their base and extend along their borders.Sometimes they become fused together into a mass,so that they project into the blood-stream in the form of an irregular funnel studded with calcareous nodules;at other times adhesions may take place between the valves with little or no increase of tissue,so that they present the appearance of a thin membrane stretching across the orifice. The most extensive rigidity and calcification of the valves will be found in old rheumatic subjects,and in cases the line of attachment of the valves to the aorta becomes entirely obliterated.Obstruction at the aortic orifice is frequently accompanied by atheromatous changes in the aorta,which becomes dilated immediately beyond the aortic ring.In some instances the contraction of the valves at their points of attachment causes them to form deep pockets or pouches behind the cusps. Sometimes the valves are studded with vegetation.As an immediate result of aortic stenosis the wall of the left ventricle becomes hypertrophied.This change is usually a gradual one,and is due to the increased force required to propel the blood through the constricted orifice.If the hypertrophy fully overcomes the obstruction to the outgoing current of blood,there will be no change in the cardiac cavities;but if the compensation is incomplete,the cavities will become dilated,the lungs congested,and the right ventricle hypertrophied. A late result is insufficiency of the mitral valves. The amount of blood which is propelled into the aorta at each ventricular systole in a well-marked case of aortic stenosis is below the normal,and consequently

the supply of blood in the arterial system is diminished and the general nutrition of the patient becomes impaired.Aortic insufficiency is produced either by an abnormal condition of the aortic valves or by dilatation of the aortic orifice,which prevents its complete closure during the cardiac diastole and allows a backwash of blood from the aorta into the left ventricle.Acute endocarditis rarely causes it,unless it isattended by extensive ulcerations,when it may lead to a rapidly fatal termination.Its most frequent cause is fibrotic endocarditis,which leads to thickening,induration,and contraftion of the valves.Such fibrotic changes are sometimes met with in rheumatic cases under conditions which lead to persistent tension of the valves during cardiac systole.In this lesion the cusps are prevented from performing their normal function on account of the following anatomical changes:As a result of chronic endocarditis the cusps may have been thickened,puckered,and shortened,so that they do not meet when brought into the plane of the orifice.When the central portion of the segment is indurated,the whole valve subsequently curls up,either towards the orifice or back against the wall of the aorta,and in either case there is insufficiency of the cusps.In the one case there is insufficiency with great obstruction:in the other,with but very slight obstruction.These processes of thickening and shortening may also result from fibrotic changes extending from the aorta to the cusps.Regurgitation may result also from adhesion of the valve tips to the walls of the aorta or the rupture of a segment of the valve. These fibrotic changes may be combined with atheroma and calcification.Insufficiency from dialatation of the aortic orifice,the result of extensive fibrotic changes in the ascending portion of the aorta,is not infrequent.During diastole,normally,the blood is passing from the auricle into the emptied ventricle;when, however,regurgitation has persisted for a considerable time,there will be added to the primary stream,which of itself is capable of filling the cavity of the ventricle, a regurgitated stream from the aorta;and by this combination of two streams the left ventricle becomes overdistended and permanently dilated.This dilatation occurs all the more readily since during the diastole the ventricular walls are relaxed and less capable of resisting the increased blood-pressure.This permanent dilatation of the left ventricle occurs in a comparatively short time,and to overcome the dilatation and the obstruction to the cardiac circulation,which allows of over-distension of the ventricular cavity,the walls of the ventricle hypertrophy.The hypertrophy goes on increasing until it compensates for the dilatation,but before this point is reached the ventricular cavity sometimes becomes very much dilated and the left heart reaches an enormous size.The dilatation,and in such cases often hypetrophy,reach their extreme limit,the heart weighing thirty or forty ounces;Fagge records a case in which its weight was forty-eight ounces.This is sometimes called bovine heart.In such a case the heart has a peculiar pointed form,the right ventricle appearing like a mere appendix.The left ventricle becomes capable of containing an abnormally large quantity of blood,so that the arteries are over-distended with each systole of the heart.The concussion caused

by the increased ventricular power forcing an abnormally large quantity of blood into the aorta causes extensive fibrotic changes in its walls, and results in dilatation. The coronary arteries become secondarily involved in the fibrotic changes, their orifices are narrowed, and the nutrition of the hypertrophied cardiac walls is consequently diminished. In the normal heart the aortic recoil is undoubtedly the principal force which propels the blood into the coronary arteries. When the aortic valves are insufficient and furnish little or no resistance to the return current of blood, we have another factor in diminishing the coronary blood-supply; therefore in all cases of aortic insufficiency with extensive dilatation and hypertrophy of the heart, degenerative changes must sooner or later take place in the muscle of the heart. In certain instances atrophy of the papillary muscles allows the mitral flaps to swing back into the left auricle when increased pressure is exerted upon them; when from any cause mitral incompetency becomes secondary to and coincident with aortic insufficiency, all the signs of impeded venous circulation will be present. When over-distension of the left ventricle causes incomplete emptying of the left auricle, a greater or less amount of passive hyperaemia of the lungs may be present without mitral insufficiency. In consequence of the increased amount of blood which is forced into them with each forcible ventricular systole, the systemic arteries undergo fibrotic changes.

Stenosis of the mitral orifice is a common consequence of rheumatic endocarditis; and the morbid conditions which occasion it are thickening and contraction of the segments of the valve, adhesion of the free edges of the valve tips, and agglutination of the chordae tendineae. The free edges of the valves are often thickened greatly, and calcareous masses may develop in the thickened portion. When the chordae tendineae and papillary muscles have become adherent, the edges of the valves are drawn down into the cavity of the heart. As the flaps are adherent along their adjacent borders, the valve presents a funnel-shaped appearance with its base looking toward the auricle, and with a slit-like opening into the ventricle below, which may scarcely admit the tip of the little finger. This has been called the buttonhole slit. Sometimes the flaps are stretched horizontally across the auriculo-ventricular ring like a diaphragm, with a small crescentic opening in the centre. In rare instances the valve segments are normal, and the stenosis results from large calcareous masses which project into the mitral opening from auriculo-ventricular ring. As a result of the mitral stenosis the left ventricle becomes smaller, and sometimes its walls become thinner than normal. The aorta also is smaller and the walls are thin. There are always dilatation and hypertrophy of the left auricle. Sometimes the auricle is enormously dilated, so much so that Thurman, more than half a century ago, described it as a true aneurism of the left auricle. Not infrequently the auricular walls measure an eighth of an inch in thickness. Its appendix is elongated, assuming a peculiar curved form, the aperture between which and the auricle proper becoming larger than normal. Sometimes it reaches two and three-quarter inches in length. For a time the auricular

hypertrophy overcomes the obstruction to the blood-current at the auriculo-ventricular opening,but gradually the compensation is broken and the pulmonary circulation becomes obstructed,causing congestion of the pulmonary veins.To overcome this the right ventricle hypertrophies,and for a time is able to counteract the pressure in the pulmonary vessels,but sooner or later dilatation occurs with incompetency at the tricuspid orifice,and congestion of the systemic veins follows. The pulmonary congestion which results from the obstruction in the pulmonary veins may lead to those changes which collectively constitute brown induration of the lung and thrombosis of the right ventricle of the heart. Mitral incompetency is due to a condition of the mitral valve which allows the blood to flow back from the left ventricle into the left auricle during the contraction of the ventricle.Rheumatic endocarditis is the primary cause of most of the changes which lead to this condition.The most common pathological changes which occasion it are thickening,induration,and shortening of the flaps from chronic endocarditis.The valve may be rendered incompetent also by contraction of the chordae tendineae,by adhesion of the valve leaflets to the ventricular wall,or by their rupture or that of the chordae tendineae.Calcareous plates at the base of the valve may present the closure of the segments of the valve,or all the structures at the mitral orifice may be converted into a firm calcareous mass.In such cases more or less obstruction accompanies the regurgitation. Mitral insufficiency due to such dilatation of the ventricle sufficiently great to render it impossible for the mitral segments to close the auriculo-ventricular orifice is seldom encountered except with aortic insufficinecy.Mitral incompetency causes dilatation of the left auricle,due to the pressure of two blood-currents during its diastole,one from the lungs and the other from the left ventricle.This dilatation is followed by thickening and hypertrophy of the left ventricle.This dilatation is followed by thickening and hypertrophy of the auricle.Following this the pulmonary circulation is impeded,the pulmonary vessels become dilated,and their walls undergo fibroid changes as the result of the continued regurgitant pressure,and the lung tissue undergoes those changes which are incident to passive congestion of the pulmonary vessels.This constant obstruction to the return circulation of the lungs interferes more or less with the outward current of blood to the lungs from the right ventricle.As the obstruction comes on gradually,the right ventricle becomes hypertrophied,so as to overcome it.Consequently the hypertrophy of the right ventricle compensates at first for the mitral insufficiency,and as long as the left ventricle is able to overcome the increased resistance in the lungs the systemic circulation is not impaired.But after a time this compensatory hypertrophy ceases,the right auricle becomes dilated and hypertrophied,the tricuspid valve becomes incompetent,and the congestion of the systemic is not impaired.But after a time this compensatory hypertrophy ceases,the right auricle becomes dilated and hypertrophied,the tricuspid valve becomes incompetent, and congestion of the systemic veins results.This impedes the hepatic and portal circulations,and is indicated by passive congestion of the abdominal viscera

and by cyanosis of the surface during active physical exercise.Not infrequently moderate mitral incompetency with considerable dilatation and hypertrophy of the left auricle and ventricle may exist for years without any disturbance of the pulmonary or systemic circulation,but there is constant danger in such cases that sudden overtaxing of the heart will break the compensation and give rise to alarming symptoms.Acute dilatation of the left ventricle,the result of disease at the aortic orifice,may cause a relative incompetency of the mitral valve,which is sometimes immediately followed by extensive dilatation of the left auricle and engorgement of the lungs to a very severe degree.

It is the rarest possible thing for the right side of the heart to be affected in rheumatic fever,such location of lesions being mostly congenital.Pulmonary stenosis rarely results from endocarditis or disease in the pulmonary artery.When it does occur,there is generally adhesion of the valve segments.Bertin records an instance of this affection where the adherent valves formed a horizontal septum across the pulmonic orifice, the opening being barely a fourth of an inch in diameter.A thickened and projecting tricuspid valve has been found to be the cause of obstruction at the pulmonic orifice,the pulmonary valves themselves being normal. Obstruction of the pulmonary artery just beyond the valves may be caused by the pressure of aneurisms, tumours of the anterior mediastinum,and enlarged bronchial glands.Lond-standing obstruction at the pulmonary orifice is followed by compensatory hypertrophy of the right ventricle,accompanied by tricuspid regurgitation and dilatation of the right auricle.Tumours may be found pressing on thepulmonary artery,diminishing its calibre. Pulmonary incompetency is a lesion of the valves of exceeding rarity;indeed,there are not a few writers who say that it never occurs.But there are a few apparently genuine cases in the literature.Being more or less of hypothetical occurrence,it need not be dpecially described.

The literature contains a few instances of tricuspid stenosis in connection with mitral stenosis.In some of the cases the tricuspid flaps were thickened and united for a third of their length;in others the valve flaps formed a diaphragm with a central opening scarcely admitting the point of theindex finger.Hypertrophy of the heart is associated with the lesion.The opening may be so small as not to admit the tip of the little finger;and it may be present along with extensive mitral stenosis and hypertrophy of both auricles and ventricles. Tricuspid incompetency may be the result of endocarditis occasioning thickening and puckering of the valve,but its most common cause is stenosis or incompetency of the mitral valve.When it is due to rheumatic endocarditis,the tricuspid valve may be thickened and retracted or adjacent flaps adherent,and the columnae carnae and papillary muscles shortened.Obstruction in the pulmonary circulation,secondary to mitral disease, causes dilatation of the right ventricle,which renders the tricuspid valve relatively insufficient.The first effect of tricuspid insufficiency is dilatation and hypertrophy of the right auricle.When the valves in the subclavian and jugular veins become unable to resist the regurgitant current of blood,jugular pulsation

follows.Before this occurs,however,the branches of the inferior vena cava and the organs to which they are distributed become engorged,for they have no valves to resist the backward flow of blood.The inferior cava and the hepatic veins sometimes become greatly distended under these circumstances,and the liver presents the peculiar appearance known as nutmeg liver.Later the skin assumes dingy-yellow hue.If cyanosis be present,a peculiar greenish tint is produced which is met with only in tricuspid incompetency.The spleen is enlarged and hard,the mucous membrane of the stomach is congested,and often presents numerous ecchymoses and haemorrhagic erosions. Intestinal catarrh develops,and the general venous congestion in the abdominal cavity causes haemorrhoids and ascites.The kidneys become congested and have a hard, stony feel.Thrombi may form in the femoral veins. The venous stasis in the lower parts of the body is followed by transudation of serum first in the ankles,and then the dropsy progresses upward until general anasarca is ultimately produced.

S Y M P T O M A T O L O G Y.

The symptomatology of acute articular rheumatism can most conveniently be considered in two sections,the first to include the general phenomena of the disease and the second the analysis of the individual phenomena including complications and sequels.

GENERAL SYMPTOMS.

In some cases of rheumatic fever,but in very few, there are prodromal symptoms lasting for a day or two, consisting of headache,lassitude,loss of appetite,a coated tongue,indigestion,constipation,muscular pains, chilliness,mild tonsillitis,pharyngitis or laryngitis, a pale,flabby,coated,and teeth-indented tongue,or epistaxis.Generally speaking,the invasion of the malady assumes one or other of three forms:either(a)slight disorder of health,such as debility,failure of appetite, unusual sensibility to atmospheric changes,dragging pains in the limbs or joints or in some muscle or fascia,which precede by one or more days the fever and general disturbance;(b)sometimes a mild rigor or repeated chilliness,accompanied or soon followed by moderate or high fever,with in a few hours to one or at most two days the characteristic articular symptoms;or(c)in very exceptional instances febrile disturbance,ushered in by chills,followed perhaps by inflammation of the endo- or pericardium or pleura before the joints are affected. No matter what may be the mode of invasion,the symptoms of the established disease are well defined,and marked febrile disturbance,transient inflammation of several of the large articulations,excessive activity of the cutaneous functions,and a great proclivity to inflammation of the endocardium and pericardium constitute the

classical phenomena of the disease.The real onset of the affection commences with the first rise of temperature,which is sometimes ushered in by a single rigor, more or less marked,sometimes by slighter and repeated attacks of shivering.The first elevation of temperature is usually moderate,not exceeding 104.F.In a general way, the rise of temperature is proportionate to the number of joints involved and the intensity of the articular inflammation.Other symptoms of fever show themselves at the same time.The head may be affected;the patient complains of thirst and loss of appetite;the pulse and breathing are quickened.These symptoms are all of moderate severity,corresponding to the not excessive rise of temperature.On the same,or at least on the following, day one or other of the joints,usually of the lower extremity,becomes painful,and,when sufficiently near the surface,hot and swollen and more or less distinctly reddened as well.The patient is afraid to move the affected limb;he feels even the slight weight of the bedclothes.Soon,moreover,we are able to detect fluctuation in the joint,when its anatomical position allows of its being examined,as,for example,in the case of the knee, elbow,or shoulder-joint.After a short interval,the fever either remaining stationary or undergoing a slight increase,fresh joints become involved,while in those originally affected the inflammation generally subsides; usually,but not always does this occur.In exceptional instances they may continue swollen and painful,or,after a short interval of health,they may become inflamed anew.In this way most of the joints of the body may be successively affected;and,when the disease is localised in the larger articulation,such as the knee or hip or intervertebral joints,the patient is in a really deplorable condition.He suffers agony when he tries to micturate,when his bowels have to be relieved,when his bed- or body-clothing is changed.He lies motionless in bed, and the slightest vibration will make him scream aloud. We not infrequently find that the pain is very acute without any corresponding severity in the other signs of articular inflammation;exacerbation frequently occurs without obvious cause,especially towards evening;the inflammation often extends from the joints to neighbouring parts,to the tendons and muscles,and perhaps even to the nerves.The affected joints often show swelling and oedema all around them.The skin does not usually feel very hot to the touch.In spite of the fever it is usually bedewed with perspiration,which has a sour odour and often raises the skin in minute vesicles or sudamina.Other forms of eruption are likewise not uncommon.The urine is scanty,concentrated,and of high specific gravity;it is stronglyacid,and soon deposits a sediment of brick-red urates and free uric acid. The tongue is moist and coated with a whitish fur;the bowels are usually confined;the faeces,even at the height of the disease,are usually more dry and hard than in health,or than they are in patients kept on the same diet, this being due to the loss of water in the perspiration. The patient's mind,except in the conditions in due course to be described,remains inclouded during the whole course of the disease.Delirium is either absent altogether,or there may be a little wandering,in specially sensitive and irritable patients,when the fever undergoes an exacerbation.Sleep is usually interfered

with by the severity of the pain;indeed,insomnia and pain in the joints are often the only symptoms of which the patient complains;occasionally the copious sweating is an additional source of annoyance to the sufferer. In this way the affection may continue for weeks,in the absence of complications,without following any typical course,its local manifestations,that is the number of joints affected and the violence of the articular inflammation,undergoing alternate exacerbation and remission,until at length no fresh joints become inflamed,the fever and sweating abate,together with the pain in the joints already affected,and recovery ensues.The articulations which have been inflamed may recover completely,or,in less favourable cases,some residue of the inflammatory process may be left in them.As a rule,acute articular rheumatism runs its course in from three to six weeks;but after recovery,the joints may remain peculiarly susceptible for a considerable period,showing a tendency to renewed inflammation,by which the process of convalescence may be easily interrupted.But even apart from such interruptions,convalescence from a severe attack of rheumatic fever is usually very slow;the patient is generally much reduced in strength by the attack he has passed through,and cardiac troubles may occur.Simple and uncomplicated cases seldom die. The fatal issue,when it does occur,is commonly preceded by a rapid elevation of temperature,with all the signs of a profound disturbance of the nervous centres,delirum and an exceedingly rapid pulse,speedily followed by coma and collapse.In certain instances the affection is not of this medium severity.Sometimes,in its earliest stages or even throughout its entire course,only a few joints are affected,and these only in a moderate degree.In such mild cases the fever and other disturbances are proportionate to the local mischief,and are very slight;the temperature,especially in the morning,scarcely rises above the normal;the appetite and sleep are but little interfered with,and the disease may subside in a comparatively short time,perhaps in from a week to a fortnight after the commencement of the attack. But it may at any moment pass into a graver form by implication of fresh joints in the inflammatory process.Other deviations from the normal type may be caused by the supervention of complications,which are more numnerous and more varied in this than in this than almost any other disease,and which readily tend to modify the course,duration,and especially the issues of rheumatic fever.Nevertheless,even the complications seldom lead to a fatal issue;but they often prevent complete recovery by leaving behind them incurable organic disease, to the effects of which the patient succombs long after the disappearance of the parent disease.

ANALYSIS OF THE SYMPTOMS.

For the complete understanding of the protean manifestations of acute rheumatic fever it is necessary to consider the more prominent and important of the individual phenomena in special detail.

JOINTS.

Should the articular phenomena not have occurred at the same time as the fever, they will follow it in from twenty-four to forty-eight hours. At first one or more joints, usually the knees or ankles, become painful, sensitive to pressure, hot, more or less swollen, and exhibiting a slight blush of redness or none at all. The swelling may consist of a mere puffiness, due to slight infiltration of the soft parts external to the joint, or of a more or less considerable tumefaction, caused by effusion into the synovial capsule. In the knees, elbows, shoulders and hips the swelling is usually confined to the articulations, and there is but little redness of the integument, but in the wrists and ankles the inflammatory process is often more severe, and may invade the whole dorsum of the hand or foot, rendering the integument tense, tumid, red, and shining. Pitting of the swollen parts, although quite exceptional in acute articular rheumatism, will exist under the conditions just mentioned. The metacarpo-phalangeal articulations are likewise often a good deal swollen and of a bright-red colour. The pain in the affected joints varies from a slight uneasiness or dull ache to excruciating agony; sometimes the pain is felt only on moving or pressing the joint; pressure almost always aggravates it; even the weight of the bedclothes may be intolerable; and in severe cases the slightest movement of the joint or a jar of the bed produces great suffering. The pain, like the swelling, sometimes extends beyond the affected joints to the tendinous sheaths, the tendons, and muscles, and even to the nerves in the affected locality. The skin over the affected joints shows increased sensibility to changes of temperature, but a diminished sensibility to faradic irritation. The shifting character of the articular affection is one of the most striking peculiarities of the rheumatic attack, the inflammation tending to invade fresh joints from day to day, usually, but not invaraibly declining in those first affected; and sometimes this retrocession of the inflammation in an articulation is so sudden, and so coincident with the invasion of a different one, that it is often regarded as a true metastasis. Exceptionally, however, one or several joints remain painful and swollen, although this occurs chiefly in subacute attacks. In this way most of the large joints may successively suffer once, twice, or oftener during an attack of acute rheumatism. And as the inflammation commonly lasts in each articulation from two to four or more days, it is usual to have six or eight of the joints affected by the end of the first week. While the ankles or knees, wrists, elbows, and shoulders, are especially liable to be affected, and with a frequency pretty closely corresponding to the above order, the joints of the hands occasionally, and hips even more frequently, escape. So far as my own observations go, the intervertebral and tempero-maxillary articulations are very exceptionally involved, the usual location of the disease in the different joints being: Foot, 27.8 per cent.; knee, 17.9 per cent.; hand, 9.6 per cent.; shoulder, 6.2 per cent.; metatarsus, 3.7 per cent.; elbow, 2.2 per cent.; metacarpus, 1.2 per cent.; toes, 0.8 per cent.; fingers, 0.8 per cent. Analyses of published cases show that the lower extremities are more apt to be involved than the upper, the figures being as follows: Knees, 65

per cent.;hands(wrist or fingers),45 per cent.;ankles, 40 per cent.;shoulders,10 per cent.Bosanquet(Lancet, June 2,1900) has analysed 450 cases of rheumatic fever treated at the Charing Cross Hospital from 1890 to 1897,and in them the knee and ankle were nearly twice as often affected as any of the other joints.

TEMPERATURE.

The temperature in cases of rheumatic fever pursues no typical course,and usually exhibits a series of exacerbations and remissions which correspond closely in time and degree with the period,duration,and severity of the local inflammatory attacks.As a very general rule in average cases,the temperature attains by the end of the first or second day the height of 102.F.,and while the subsequent evening exacerbations may reach 104.,104.4.F., or very rarely 105.,yet in the great majority the maximum temperature does not exceed 103.F.,and in a very considerable number falls short of 102. An analysis of one of Southey's tables(St.Barthol.Hosp. Reps.,xiv,p.12)shows that in eighty-four cases of acute rheumatism one attained the temperature of 105.8.;eight that of 104.to 105.;fifteen that of 103.to 104.;thirty-two that of 102.to 103.;seventeen that of 101.to102.; ten that of 100.to101.;and one that of 99.8.;that is, the temperature was below 103.in five-sevenths,and below 104.in about ten-twelfths,of the whole.In very mild cases,in which but a few joints are inflamed,and only to a slight degree,the temperature may not reach 100.at any time,and there may be intervals of complete apyrexia.On the other hand,in a few rare severe cases of rheumatic fever,especially when complications with pericarditis,pneumonia,or delirium,or other disturbance of the cerebral functions,the temperature attains to 106.,108.,109.4.,110.2.,or even 111.,or 112.F.Such cases are spoken of as examples of rheumatic hyperpyrexia.There is no rule about the mode of invasion of this high temperature.It may come on gradually or suddenly, the previous range being low,moderate,or high,steady or oscillating.Hyperpyrexia is a well-known occasional symptom in this disease,and it sometimes rises exceptionally high suddenly and without known cause.Such cases exhibit usually delirium,a feeble pulse,sweating,and become alarmingly ill;but Da Costa reported a temperature of 110.F.without delirium.Ringer lost a case with the same temperature,and Fox reported a recovery after a temperature above 110.F.,which was treated with cold baths;another case met with fatal issue at 112.F.Newman(lancet,June 16,1900)reports the case of a woman of thirty years.On the twenty-first day of the disease the temperature rose to 107.8.F.,the pulse being 174,weak,and irregular.This continued about twenty-four hours.Sponging with water at 48.F.reduced the temperature in that time,and the thermometer subsequently did not register above 102.F.A soft mitral systolic murmur was heard.Patients with rheumatic hyperpyrexia are apt to die in coma.The temperature may continue to rise after death.Defervescence in acute articular rheumatism takes place,as a very general rule,gradually,that is by lysis;but exceptionally it is completed in forty-eight or even twenty-four hours.An interesting observation,which if reliable will be of great prognostic value,has been made by Southey(St.Barthol.Hosp.Reps.,xiv,p.16),to the effect that a "short period of defervescence,or a

sudden remission and an early remission, betokens the relapsing form of the disease, and the likelihood of frequent relapses, as well as of slow ultimate recovery, in the direct ratio as this defervescence has been early and abrupt!

PERSPIRATION.

One of the most constant and remarkble accompaniments of the rheumatic attack is the occurrence of a profuse, very acid, and sour-smelling perspiration. For a long time it was generally believed to indicate an excessive formation in, and elimination of acid from, the system, either lactic acid or some of the acids normal to the perspiration, as butyric, acetic, and formic. Sir William Gull effectually upset the popular belief that the acid perspiration was Nature's method of ridding the system of too much acidity by remarking that the sweat may be both alkaline and acid in different parts of the body simultaneously, the acid reaction depending upon sebaceous decomposition. Not only have other observers failed to detect lactic acid in the perspiration of acute articular rheumatism, but demonstrated that the excessive acidity of the perspiration in this disease is but very partiallydue to the perspiration itself, and is chiefly owing to chemical changes taking place in the overheated and macterated surface of the skin and its epidermis, and to the retention of solid products accumulated on that surface. Besnier says that if in acute articular rheumatism, or other disease attended with much perspiration the surface be kept well washed, the sweat will be found in the greater number of cases at the moment of its secretion to be nearly neutral as soon as actual diaphoresis occurs, more decidedly acid when the perspiration is less abundant or begins to flow, and exceptionally alkaline. Most practitioners are aware that the profuse perspiration of rheumatic fever are non-alleviating; it is not a real critical discharge of noxious materials from the system, nor is it followed by prompt reduction of the temperature and other symptoms. It is but a symptom of the disease, and occurs especially in severe cases, and when it continues long after the reduction of the temperature is a source of exhaustion, and may be checked with advantage.

SKIN.

The profuse perspiration in this disease gives rise sometimes to widespread sudamina and miliary vesicles, in addition to which several other cutaneous manifestations are observed, such as especially erythema marginatum, erythema papulosum, and erythema nodosum. A well-marked urticaria frequently precedes acute rheumatism in certain instances; it may occur during its course or soon after the cessation of the pains. Scarlatiniform eruptions are occasionally observed, and very rarely punctiform haemorrhages - peliosus rheumatica or rheumatic purpura. The purpuric symptom may be accompanied by erythema or urticaria, and may precede, accompany, or laternate with other rheumatic manifestations. Unlike purpura variolosa and idiopathic purpura haemorrhagica, this variety is benign. In addition to a slight local oedema affecting the malleoli, scrotum, eyelids, etc., or accompanying the cutaneous eruptions just mentioned, a more decided infiltration of the subcutaneous cellular tissue occasionally exists in the vicinity of the inflamed joints and tendinous sheaths, and more

rarely extends to an entire limb, which may or may not be considerably enlarged and painful and resemble a milk-leg, but may be red, hot, and tender, and excite suspicion of phlegmonous erysipelas. Phlebitis, although infinitely less frequent than in gout, has been observed in rheumatic fever. Jaccoud (Pathol. Interne, ii, 1871, 546), in 1871, mentioned the exceptional occurrence of subcutaneous nodosities in rheumatism, which he says Froriep first pointed out; but Homolle (Rev. de Méd., T. i, 1881, 297-308) states that they had been previously mentioned by Sauvage and Chomel. Since then several independent observers have met with this affection, and Barlow and Warner (Trans. Internat. Med. Congr., Lond., Vol. iv, pp. 116-128, 1881) published a monograph upon the subject based upon twenty-seven cases which they had separately or conjointly investigated. These nodules may vary in number from one to fifty, and in size from that of a pinhead to an almond, and are quite subcutaneous, firm, elastic, painless, and freely movable. They are not usually attached to the skin, but to the tendons, deep fasciae, pericranium, periosteum, etc.; the integument over them is free from heat, redness, and infiltration, although exceptionally tenderness on pressure and slight redness may exist over them. They are found most frequently on the back of the elbow, the malleoli, and the margins of the patella, but occur occasionally on the extensor tendons of the hand and foot, the scapular spine and iliac crest, the temporal ridge and superior occipital curved line, the ear, etc. These nodules occur singly or in clusters, and are often symmetrical; they are very rapidly developed in crops or in succession, and last sometimes for a few hours, more frequently from three or four days to four or five months, or even eighteen to thirty months. The original formations may disappear, and be succeeded by fresh ones; and sometimes, when no longer perceptible by touch, they may be found post-mortem. Their development is unattended by pyrexia, unless pleuritis, pericarditis, or other condition coexist to which the pyrexia might be referred. These nodosities do not appear to suppurate or ossify or become infiltrated with urate of soda, and histologically they resemble organising granulation tissue. As regards their pathological associations, Barlow and Warner found evidences of rheumatism in twenty-five out of twenty-sen cases; a morbid condition of the heart existed in all of them, and chorea in ten of them. Two of the conclusions formulated by the authors just mentioned are of importance: that these subcutaneous nodosities may be considered as in themselves indicative of rheumatism, even in the absence of pain; that, while unimportant in themselves, they are of serious import, because in several of their cases the associated heart disease was found actively progressive. Duckworth reported two cases in which these nodules occurred in adults, lasted eighteen months in one, and were still present in the other case after thirty months, and were attached to the skin and periosteum. In one of them the nodules were very painful and ached more in cold weather, and the patient had no history of rheumatism or of chorea, although her mother and one sister had (Brit. Med. Jour., i, 1883, 868). Stephen Mackenzie (Ibid., i, 1883, 867) mentions the case of a woman - family history not given - who was the subject of syphilis in its tertiary stage; there was neither rheumatism nor chorea in her history, and she was free from disease of the heart.

SALIVA.

The normally-alkaline saliva has generally a strong acid reaction in rheumatic fever, and, according to Fenwick, it always in this disease contains a great excess of the sulphocyanides, which slowly and steadily diminish until they become normal in amount at the end of the third week. The patient complains of great thirst, and the mouth is dry and parched.

KIDNEYS.

The urine presents the usual characteristics of the febrile state. It is diminished in amount, is of a red-brown colour, high specific gravity, and strong acidity. It is loaded with urates, and often with uric-acid crystals, but the chlorides, as in other fevers, are notably diminished or wholly absent while the fever persists. There is often a temporary trace of albuminuria, with a few hyaline or granular casts, but a serious nephritis has sometimes been observed. It is only comparatively seldom that the kidneys become diseased in acute articular rheumatism, if we except embolism of their arteries due to endocarditis; and, according to some, it is very doubtful whether an acute parenchymatous nephritis as observed in rare instances can be referred to direct rheumatic inflammation or not, but to the operation of the exposure which influenced the occurrence of the rheumatic fever.

BLOOD.

A condition of excessive coagulability of the fibrin, which, according to various authors presents a much increased formation([redacted]), is a constant feature of acute articular rheumatism; however, in very serious cases, especially those attended by hyperpyrexia and grave cerebral symptoms, the blood after death has been black and coagulated, and the fluid in the serous cavities has given an acid reaction. These and the other alterations in the blood usually are proportionate to the intensity of the fever and the number of the joints and viscera involved. The occurrence of anaemia in this disease is well known. In many cases of average severity the loss of red blood-corpuscles is slight, while the haemoglobin falls to sixty-five or eighty per cent. In more severe cases Hayem seldom found more than four million cells; Sorensen found an average of a fraction over that number in eight cases. In prolonged and relapsing cases the red cells not infrequently fall slightly below four millions. Yet in a few of the published cases are the examinations sufficiently extensive to show that such anaemia is referable to the acute disease. Turk, while admitting the usual presence of anaemia even during the febrile period, saw a distinct increase in red cells beginning with defervescence, and in Cabot's case it does not appear either that there was any uniform loss of red cells or that the cases of long standing were invariably the more anaemic. It seems, therefore, that the severity of the anaemia following rheumatic fever has been made rather too much of. The haemoglobin in acute articular rheumatic cases suffers more severely and more constantly than the red blood-corpuscles. Cabot's average in thirty-one cases was sixty-seven per cent., and a few cases showed that it fell below sixty per cent., while Turk found between sixty and eighty per cent. Both Cabot and Lichtenstern state that the prompt administration of the salicylates checked the loss of

haemoglobin in their cases.In convalescence the restoration of the haemoglobin remains considerably behind that of the red blood-corpuscles.According to Rieder and others,mild cases without exudation usually present no distinct leucocytosis.When fever and swelling of joints exists there is always an increase to ten or fifteen thousand cells.Turk maintains that when the leucocytes reach twenty thousand or more there are nearly always complications,such as pleurisy,pericarditis,or pneumonia.Hayem also found seventeen to eighteen thousand cells in moderately severe and cerebral types of the disease. Cabot reports twenty-one to thirty-one thousand cells in six cases,one complicated with acute endocarditis, while in the others there were no complications. Other authors support his statement,having found signs of pneumonia or pericarditis,or hyperpyrexia,whenever the leucocytes rose above twenty thousand.The pneumonic signs,however,are not always those of complete consolidation of the lungs.Forthwith on the occurrence of defervescence the leucocytes begin to fall to normal,and in relapses are not nearly so affected as in the intial attacks of the disease.When the leucocytes do not greatly exceed ten thousand,the proportions of the various forms are not much altered.With distinct leucocytosis the proportion of polynuclear cells rises to a considerable height.Eosins are absent only in the early stages;later,in spite of fever and exudation,they are always present in moderate numbers,while after defervvescence most cases show a distinct tendency towards eosinophilia.In one of Turk's cases there were thirteen per cent,of eosins,and in another eight per cent.,shortly after defervescence.He thinks that a high proportion of eosinophile cells during the febrile period is a favourable prognostic sign,and occurs chiefly in cases of a sel-limiting character.In one of Zappert's cases a relapse was attended with a further increase of eosins.Sittmann says that he has frequently obtained pathogenic bacteria from the circulating blood;but these cases have later been classed as examples of septicaemia and not of articular rheumatism.The principle bacteriological studies of the blood of rheumatic fever are those of Sittmann,Singer,Kraus,and Kuhnau.The first-mentioned of these observers obtained negative results from repeated cultures in five cases,and Kraus in twelve cases.Singer conducted an elaborate investigation into the condition of the blood in sixty cases,but while he found the staphylococcus albus in several instances, this germ was probably a contamination from the skin. In one case the streptococcus pyogenes was isolated,but the history shows the patient to have been suffering from inflammation of the endocardium of the heart and haemorrhagic septicaemia.Even more conclusive were the entirely negative results obtained by Kuhnau in sixty-seven cases representing all types of the disease and complications of all sorts.It is therefore my opinion that the blood in rheumatic fever patients is sterile. I have noticed that the anaemia which is so striking a feature sometimes of this disease,especially when several joints are severely inflamed,coexists very often with a systolic basic murmur,which is most often louder over the pulmonary artery, in the second left intercostal space and more or less to the left of the sternum, than over the aorta. The murmur may appear

early in the disease,but sets in most frequently when the malady is subsiding.When thus appearing late in a case accompanied by endocarditis and pulmonary congestion,it is of favourable omen and indicates improvement in the thoracic affection.A great many authorities,however,are inclined to believe,respecting the so-called anaemic murmurs,that they depend chiefly upon regurgitation through the tricuspid orifice,although Russell (Brit.Med.Jour.,1883,i,1065)and others refer them to pressure of a distended left auricle upon the pulmonary artery.

HEART.

In cases of acute articular rheumatism the pulse is increased to about 100 beats per minute or over;but it is soft and full,and when cardiac and other complications arise it shows special characteristics which will presently be described.In exceptional cases it is very rapid,feeble,and irregular,apart from the influence of the cardiac involvement.

Great importance attaches to the cardiac affections that so frequently accompany acute articular rheumatism;they may arise in any case,even the mildest,or at any stage of the disease,and consequently the careful examination of the heart must not be overlooked in even the most apparently trivial rheumatic attack.Indeed,by some the cardiac affections are regarded as integral elements of the disease,for they occur in a large proportion of the cases,often coincidentally with the articular affection,and may even precede it,and probably may be the sole obvious manifestation of acute rheumatism,although under the last-mentioned circumstances it is not easy to establish the rheumatic element of the attack.Heart affections in this disease may be divided into inflammatory and non-inflammatory.The former include pericarditis,endocarditis,and myocarditis;the latter embrace deposition of fibrin on the valves,and the formation of thrombi,temporary incompetence of the mitral or tricuspid valves,and the formation of thrombi in the cavities of the heart.Absolute reliance cannot be placed on the statements of authors regarding the gross frequency of recent cardiac affections in rheumatic fever;for not only do reporters differ widely on this point,but they do not distinguish recent from old disease,nor inflammatory from non-inflammatory affections, nor haemic from organic murmurs.Nor does it appear probable,from the innumerable statistical compilations,that these differences are owing to peculiarities of country or race.The gross proportion of heart disease of recent origin in acute and subacute articular rheumatism was in Fuller's(On Rheumatism,Rheumatic Gout,etc.,Ed.3,p. 280)cases thirty-four per cent.;in Peacock's(St.Thomas' Hosp.Reps.,Vol.x,p.19)thirty-two per cent.;in Sibson's (Reynolds' System of Med.,Vol.iv,186),omitting his threatening or probable cases,fifty-two per cent.; in three thousand,five hundred and fifty-two St Bartholomew's Hospital cases analysed by Southey(loc.cit.,264) twenty-nine per cent.;in Bouilland's cases,quoted by Fuller(loc.cit.),five per cent.;in Lebert's(loc.cit.) twenty-three per cent.;in Vogel's(loc.cit.)fifty per cent.;in Wunderlich's(loc.cit.)twenty-six per cent.Other published statistics show a still higher percentage of cardiac involvement in this disease.Various circumstances influence this frequency.Some unexplained influence,

such as is implied in the terms endemic and epidemic constitution,appears to obtain.Peacock found the proportion of cardiac complications in rheumatism to range from sisteen to forty per cent.during an observation extending over five years,and a similar variability is shown in Southey's statistical table covering eleven years.It may be noted that these variations occurred in the same hospitals and under,it may be presumed,very similar conditions of hygiene and therapeutics.Youth predisposes to rheumatic inflammation of the heart,so that it may still be said that the younger the patient the greater the proclivity.Of Fuller's cases fifty-eight per cent.were under twenty-one,and the liability diminished very markedly after thirty.Of Sibson's cases sixty-two per cent.were under twenty-one.In infancy and early childhood the liability is very great,and at those periods of life the heart,and more especially the endocardium,rarely escapes;and the cardiac inflammation often precedes by one or two days the articular.The careful observations of Sibson confirm the spirit,but not the letter,of Bouilland's original statement,and proves that the danger of heart disease is greater in severe than in mild cases of rheumatic fever,and this is especially teue of pericarditis;the number of joints affected is very generally in proportion to the severity of the attacks.The mildest case of subacute rheumatism is not,however,immune from cardiac inflammation,and it has occasionally been observed even in primary chronic rheumatism(Raynaud,-New Dict.of Med.& Surg.,Vol.viii, 367).Occupations involving hard bodily labour or fatigue,whether in indoor or outdoor service,render the heart very liable to rheumatic inflammation.Existing valvular disease,the result of a previous attack of rheumatism,favours the occurrence of endocarditis in that disease.Some authorities maintain that treatment modifies the liability to rheumatic affection of the heart,and this will be discussed hereinafter.The period of the rheumatic fever at which cardiac inflammation sets in varies very much,but it may be confidently stated that it occurs most frequently in the first and second weeks,not infrequently in the third week,seldom in the fourth,and very exceptionally after that,although it has happened in the seventh.An analysis of Fuller's (loc.cit.,pp.77-278)experience in twenty-two cases of rheumatic fever and fifty-six of endocarditis,a total of seventy-eight,shows that the disease declared itself under the sixth day in eight;from the sixth to the tenth in twenty-nine;from the tenth to the fifteenth in seventeen;from the fifteenth to the twenty-fifth in eighteen;and after the twenty-fifth in six.The friction sound was audible in Sibson's cases of rheumatic pericarditis from the third to the sixth day in ten,and before the eleventh day in thirty,or nearly one-half of the whole.He concludes that in a certain small proportion of the cases,amounting to one-eighth of the whole, the cardiac inflammation took place at the very commencement of the disease,and simultaneously with the incarion of the articulations.

Endocarditis is the most frequent of all the forms of heart disease occurring in rheumatic fever,and in a large proportion of cases it may exist alone;pericarditis is also very often observed,but it is seldom found by itself,being in the vast majority of instances

combined with endocarditis and occasionally with myocarditis.It is usually the ordinary verrucose endocarditis that obtains.The ulcerative form occurs sometimes, and should be suspected if in a mild or protracted case of acute rheumatism endocarditis sets in with,or is accompanied by,rigors and the general symptoms are of pyaemic or typhoid character or both,even although an endocardial murmur is not present,for extensive vegetating ulcerative endocarditis frequently exists without audible murmur. It is remarkable,as Osler(Trans.Internat. Med.Congr.,Vol.i,341)has shown,how few instances of ulcerative endocarditis developing during the course of acute rheumatism are reported;and it may be added that by no means all of these were examples of first attacks, chronic valvular lesions,the consequence of former illness,existing in many of them at the time of the final acute attack.Southey's(Trans.Clin.Soc.,xiii,227)patient, and both of Bristowe's(Brit.Med.Jour.,1880,i,798),had had previous rheumatic seizures.However,Peabody's(Med. Rec.,Sept.24,1881,361)case,one of Ross' three cases (Canada Med.& Surg.Jour.,1882,Vol.xi,1;and Ibid.,1881, Vol.ix,673),and Pollock's(Lancet,1882,ii,976)case appear to have been examples of ulcerative endocarditis occurring during a first attack of rheumatic fever.The united and thickened condition of two segments of the aortic valve in one of Ross' cases indicates old-standing disease,although no history of former rheumatism is given.Goodhart(Trans.Path.Soc.,xxxiii,52)has insisted upon the tendency of ulcerative endocarditis to appear in groups or epidemics,but the proof of this is not convincing.

Pericarditis is very commonly a manifestation of acute articular rheumatism,and is especially liable to occur in the severer types of the disease.The age at which this secondary form of the disease is met with most commonly is from twenty to thirty years.Cases have been recorded in which the pericarditis preceded the articular manifestations by several days,though they usually follow them.Rheumatic pericariditis is more common in men than women,probably because men are mor subject to rheumatism.It generally occurs with the first attack,and is seldom associated with rheumatism which has become chronic or in which the acute phenomena have subsided.The symptoms of pericarditis may be so slight that they may be easily overlooked.This is dependent to a great extent on the varying intensity of the inflammatory action,and whether it is limited to a portion or affects the whole of the surface of the pericardium.In cases which are the precursors of rheumatic fever the onset of pericarditis may be marked by rigors or a distinct chill,accompanied by a rise in temperature.There are malaise,anorexia,and perhaps headache and giddiness.The cardiac action becomes excited,and there is a tendency to palpitation.The pulse is full at first,and its frequency is increased. The respiration is hurried in accordance with the change in the pulse-rate.On the other hand there may be no symptoms whatever to indicate the development of this disease.Its invasion is insidious,or attention may be first drawn to the heart by a sense of uneasiness in the praecordial region or of constriction around the chest.Temperature is not a suggestive feature of the disease.It is influenced more by the rheumatism from

which it springs.Charcot and Lorain state that the inception of pericarditis in the aged is often indicated by a distinct subnormal lowering of the body temperature;but hyperpyrexia is otherwise very commonly observed.Pain of a dull,aching,or sharp,lancinating character may be the first symptom manifested.It is often comparable to the pain of pleurisy,but is referred to the shoulder and down the left arm.The pain,however,is by no means of constant occurrence.Associated or not with the pain there may be praecordial or epigastric tenderness.Pressure on the ensiform cartilage especially is likely to cause suffering or increase it.The pain is sometimes increased by inspiration.This is due to the movement of the diaphragm,and leads to shallow,hurried respiratory acts of the superior costal type.As the liquid effusion accumulates the severity of the pain diminishes.The cardiac action becomes turbulent,and the pulse usually becomes small and irregular both in frequency and force.It may assume the characters of pulsus paradoxicus,becoming weak or disappearing altogether with inspiration.The embarrassment of the heart's action by the effused liquid develops a tendency to syncope on exertion or emotional excitement;hence the patient avoids any movement.Generally he lies upon his back with the head slightly raised,or he may incline to the left side.In some instances the embarrassment of the circulation is so great as to induce orthopnoea.Dyspnoea is a common and important symptom.The expression of the patient is anxious,the face is dusky,and he feels a sense of impending danger.As the cardiac failure increases,the venous circulation becomes more embarrassed, the cyanosis more marked,and a serous effusion is likely to occur in the abdominal and pleural cavities.General anasarca results in some cases,but more commonly the infiltration is confined to the extremities.Amongst the more prominent pressure effects are distension of the veins of the neck(there may be a venous pulse),dysphagia, and an irritative cough.Compression of the left lung increases the dyspnoea.Aphonia is present occasionally from compression of the recurrent laryngeal nerve. The onset of pericarditis may be announced by disturbances of the nervous system,or these may occur later in the disease.In mild cases there may be only headache,dizziness,and perhaps restlessness,while in severe cases there may be sleeplessness or delirium,which may be low and muttering or violent,requiring restraint.Melancholia with an inclination to suicide may superwene.Severe nervous symptoms are especially likely to occur when there is hyperpyrexia,and are probably due to the rheumatic fever,rather than to the inflammation of the pericardium itself.In view of the fact that the phenomena of the condition are so vague and indefinite,it is necessary to carefully study the physical signs in order to establish the presence of the disease;but it must be borne in mind that not every case of pericarditis is sufficiently acute or diffused to be recognised clinically,and that many cases do not progress to the formation of large effusions.It is only in more or less typical cases of pericardial inflammation that the classical physical signs are elicited.For convenience of description it is useful to divide it arbitrarily into three stages - the dry or exudative,the

effusive, and the absorptive. Inspection of the chest gives little information until the first stage is passed, unless it be the violent beating of the heart against the chest wall, and even after the stage of effusion has been reached the amount of information to be derived depends upon the elasticity of the ribs and their cartilages. In children and young subjects distension of the pericardium shows itself by an arching forward of the precordium and its neighbourhood, with widening of the intercostal spaces, which may extend from the second to the sixth rib. There will be a restriction of the respiratory movements in this region, and if the effusion be large the whole of the left chest may remain stationary. There may be a prominence of the epigastrium from pressure upon the left lobe of the liver. If the chest wall has lost its elasticity or if old pleuritic adhesions are present binding it down, there may be only widening or bulging of the intercostal spaces in the praecordial region, or there may be no evidence at all upon inspection. Sometimes the apex beat is carried to the left and upward from its normal position, or a wavy motion may be visible over the heart. This lifting of the impulse is effected without any alteration in the relative position of the heart, and is simply a mechanical phenomenon, the accumulated liquid forcing the tip of the heart back from the chest wall, while a portion nearer the base strikes it. As absorption sets in, the chest will be seen to regain its normal shape, and there will be a return of the respiratory movements. Oedema of the chest wall seldom occurs independently of more or less general anasarca. In the early stage of pericarditis the excitement of the heart is easily detected by applying the hand to the chest. A friction fremitus may be felt. The distinctness of the fremitus will vary with change in the position of the patient, being most marked when the body is bent forwards. This occurs because the heart has a greater specific gravity than the liquid by which it is surrounded, and tends to sink. The presence or absence of tenderness in the praecordial or epigastric region may be determined by palpation. As the liquid accumulates the fremitus will become less and less apparent. If the effusion be large, it will become imperceptible. Shifting of the body from side to side will cause a change in the situation of the apex beat. Occasionally an undulatory motion will be communicated to the hand even when not visible. Palpation may be of service in determining the shape of the pericardium and its degree of distension. With absorption of the liquid effusion and reapposition of the roughened surfaces the friction fremitus will be evident again; the apex beat will become perceptible, will gradually assume its normal characters, and return to its former position. The area of praecordial dulness is found on percussion to be unaltered at first, but as the liquid accumulates it is increased both vertically and laterally. Its shape is that of the pericardial sac, irregularly conical, with its base on a line a little above the ensiform cartilage. Rotch and Ebstein have called attention to the importance of dulness in the fifth intercostal space to the right of the sternum in the early recognition of pericardial effusion. According to their observations, dulness first appears at this point, which Ebstein calls the cardio-hepatic angle, and is quite or nearly

absolute,whereas the liver dulness in health is relative. Increase in the quantity of effused liquid causes a corresponding increase in the area of precordial dulness. It may extend at times from the second rib or even the clavicle to the ensiform cartilage in the vertical direction,and from nipple to nipple laterally.The dulness extends to the right of the sternum in all large effusions.Pins says that in the pericarditis of very young children with only a moderate amount of effusion there is dulness over the left half of the back,which disappears in the genu-pectoral position.He attributes this to the relatively large heart and small chest of young children,and attaches much diagnostic significance to it.Auscultation affords the first positive indication of pericarditis,and it is during the first stages of the disease that this sign is of greatest value.Hence an early diagnosis may be made in the majority of cases. If the ear of the stethescope be applied over the pericardium,friction sounds,produced by contact of the roughened pericardial surfaces,will be heard.They are rubbing,grazing,or creaking in character,compared at times to the creaking of new leather.The sounds are superficial,and are restricted to the praecordial region. There may be only a single sound,especially when the inflammation is circumscribed and in the region of the great vessels at the base of the heart.When near the apex it is likely to be double and to possess the to-and-fro character ascribed to it by Watson.The friction sounds may be synchronous with the heart sounds or occur independently of them.As a rule,they will be heard with greatest intensity at the junction of the fourth rib with the sternum on the left side,but occasionally they are present only over the base of the heart.Under such circumstances the inflammation is limited to the neighbourhood of the great vessels.The intensity of the sounds will be increased by bending the body forward,by a full inspiration,and by pressure upon the praecordium. It is affected by bringing the percardial surfaces into closer contact.The fibrinous exudation may be abundant, and yet from softness of the fibrin or from weakness of the heart muscle no sounds will be audible.The heart sounds are normal or the second sound may be accentuated. As the liquid accumulates the friction sounds become less and less distinct,until they finally disappear.The respiratory murmur is no longer heard over the praecordial space,and the heart sounds become muffled and indistinct.The muffling of the heart sounds is very important in effecting a recognition of the disease. They become indistinct from the apex upwards,but,according to Da Costa,the second sound rarely ever disappears.As absorption proceeds the heart sounds regain their normal characters and the friction sounds reappear.They remain until the disappearance of the fibrin or until adhesion takes place between the two pericardial surfaces.The sequals of pericarditis relate mainly to the anatomical changes in the heart wall and structures surrounding the pericardium which are the direct or indirect result of the morbid process.When there has been a mild grade of inflammation the parenchymatous degeneration of the superficial layers of the heart muscle is quickly repaired.In severe cases the weakening of the muscle leads to dilatation,with sunsequent hypertrophy of varying degrees,or of the inflammation

is long continued there may be consecutive tissue increase, crowding out of the muscle fibres, and a resulting cardiac fibrosis of greater or less extent. Circumscribed adhesions are harmless. Occasionally the pericardial cavity becomes obliterated from extensive adhesions. It is stated by some authorities that the heart is always left in a weakened condition by an attack of pericarditis. If the latter be long continued, the heart may be so weakened that venous stasis ensues, with its train of symptoms. Congestion of the liver, stomach, spleen, and kidneys occur. The amount of urine decreases. Hydrothorax and general anasarca may be seen. It must be remembered that cases of pericarditis differ greatly in extent, duration, and severity. There are those in which the inflammatory action is confined to a limited portion of the pericardial surface, and when so limited the base of the heart is the seat of election; it may be that the great vessels alone are affected; or the inflammation may involve only the apex. The diagnosis of pericarditis depends upon its physical signs. The diagnosis cannot be made from the symptoms alone. It is therefore necessary to examine the heart frequently. Rigors, or a distinct chill, followed by pain in the praecordial region, should direct attention at once to the heart. If the pain is severe, it may radiate down the left arm. It is increased by pressure and sometimes by a full inspiration. Instead of distinct pain there may be only a sense of uneasiness in the preaecordium. Pain is commonly regarded as the most important symptom in pericarditis, yet it may be entirely absent throughout the course of the disease. Hyperpyrexia usually ushers in pericarditis in rheumatic fever, though there may be a distinct lowering of the temperature. The onset of pericarditis may be so insidious that there will be no symptom to draw attention to the heart until effusion occurs and produces dyspnoea or embarrassment of the circulation. The symptoms may be referred entirely to the nervous system. Headache and restlessness occur in the milder cases, while in the severer ones there will be evidence of intense cerebral disturbance. Delirium, low or muttering or severe and requiring restraint, frequently appears. Melancholia with suicidal tendencies comes on at times, and may last from two weeks to as many months. The importance of dulness in the fifth right intercostal space in the early recognition of pericardial effusion must be remembered. Later on the shape of the area of praecordial dulness, the absence of or slight change in the outline in the recumbent position, and the fact that the dulness does not extend to the back are presumptive signs of pericarditis. In young children however, and in cases with large effusions, there may be dulness over the left back. Under such circumstances other signs and such symptoms as may be present must be taken into consideration. Pins states that in children this posterior dulness is due to atelectasis at the base of the left lung, and disappears in the knee-chest position. Pericarditis frequently complicates pneumonia and pleurisy, and the praecordial may be continuous with the lung dulness. If the anterior lobes of the lung are bound to the chest wall, there may be but little change in the normal area of praecordial dulness even with large effusions. Pulmonary emphysema sometimes diminishes the area of praecordial dulness and may lead to a mistaken

diagnosis.On the other hand,consolidation of the anterior borders of the lung may give rise to dulness which it will be difficult to distinguish from pericardial effusion.The presence of a friction sound which is superficial and limited to the region of the heart is diagnostic of pericarditis if the sound continues when the respiration is voluntarily suspended.This statement, however,will not hold good for those rare pleuropericardial sounds produced by the movements of the heart against a roughened pleura.It will be impossible to make a diagnosis in these cases unless there be intermission of the sound during some of the heart beats.The friction sound in pericarditis is often limited to the base of the heart,and may continue after a moderately large effusion has occurred.Pericardial friction sounds become indistinct from the apex upwards as the effusion increases.Being dependent upon separation of the two layers of the pericardium they never disappear suddenly, though they may be heard at one visit and not at the next,so rapidly does the liquid accumulate in certain instances.Again,they may change their seat and character during the visit of the medical attendant.As the effusion increases in quantity the heart sounds become muffled and indistinct.This muffling of the heart sounds is an important aid in diagnosis,and is most apparent at the apex.It is only the first stage of a pericarditis which at times simulates endocarditis.As soon as the effusion occurs there can be no doubt,though it must be remembered that the two affections may coexist.In ordinary cases the friction sounds of pericarditis may be distinguished from endocardial murmurs by their superficial character,by their rougher quality,and by their limitation to the praecordial area.Pericardial frction sounds have a rough,grating quality as a rule,and appear to be immediately under the ear.Endocardial murmurs are soft and blowing.Yet pericardial sounds sometimes possess these qualities.In such cases the differential diagnosis is out of the question.Pericardial sounds are confined to the preacordial area,occasionally to a definite portion of it,and have their greatest intensity at the junction of the fourth rib with the sternum on the left side.Endocardial murmurs are conveyed beyond the limits of the praecordium,to the right or left, along the course of the vessels in the neck,and sometimes to the back.A praecordial friction sound may change its seat or character,while an endocardial murmur never does.The intensity of a pericardial friction sound may be altered by change in the position of the patient. Bending him forward will increase the intensity by bringing the pericardial surfaces into closer contact, while bending him backwards will diminish it.Moreover, the intensity may be increased by a full inspiration. Endocardial murmurs are not thus affected;they are often decreased by full inspiration.Pericardial friction sounds bear no definite relation in time to the heart sounds.They may be double or,as Osler informs us, passess a canter rhythm.Endocardial murmurs precede, take the place of,or follow the heart sounds.Often they entirely mask them.Pleurisy sometimes simulates pericarditis,not only in its symptoms,but in its physical signs.The dry,irritative cough and dyspnoea occur in both diseases,but the physical signs differ in location.When pericarditis complicates pleurisy or

pneumonia it is often overlooked.Pleurisy does not give rise to bulging in the praecordium.The friction sound of pleurisy is likely to be confounded with that of pericarditis only when it is confined to the praecordial region.In such cases voluntary suspension of respiration will cause the pleuritic sound to cease. The difficulty of distinguishing a pericardial from a pleuropericardial friction sound has been referred to.If the sound intermits during one or two beats of the heart,it may be considered of pleuritic origin.The shape of the praecordial dulness,and the fact that except in large effusions it does not extend beyond the praecordium, will serve to distinguish the two diseases.In pleurisy dulness is present over the whole of the left chest, being most marked in the back.If there be dulness posteriorly in pericarditis,it may disappear in the knee-chest position.Perfect distinctness of the heart sounds in pleurisy is another point in diagnosis.Hypertrophy of the heart may be mistaken for pericarditis in the stage of effusion,because of the increased area of praecordial dulness:yet a careful inquiry into the physical signs will suffice to differentiate the two conditions. In hypertrophy the force of the impulse is increased,and, if it is displaced,it is carried to the left and downwards.In pericarditis the force of the impulse is diminished,and it is displaced upward and to the left. In pericarditis the abnormal area of dulness extends to the left of the apex beat,often as much as two inches,which is never the case in hypertrophy.Such an extension of dulness beyond the apex beat may be considered diagnostic of pericarditis.In hypertrophy the heart sounds are intensified,while in pericarditis they are muffled and indistinct,and may even be inaudible at the apex. If dilatation coexists with the hypertrophy,the diagnosis may present greater difficulty.The impulse is wavy and diffused in dilatation,the area of dulness is more or less quadrilateral rather than triangular as in pericarditis,and there is only a relative dulness in the fifth right interspace.When the dilatation is advanced and the heart sounds are weak,it may be impossible to make a differential diagnosis between dilatation and pericarditis.Tumours of the mediastinum sometimes simulate pericarditis.Cases have been recorded in which the heart was enveloped in a neoplasm which filled and distended the pericardial sac without altering its shape.In such cases it is impossible to effect a differential diagnosis.New growths in the mediastinum may give rise to praecordial dulness,may cause displacement of the apex beat and interference with the heart's action, and may cause pressure effects upon the surrounding structures.But the dulness which they produce is not uniform in outline,and varies with the situation of the tumour;moreover,the area of dulness is rarely triangular,such as we find in pericarditis.The displacement of the apex beat varies in different cases,and if a solid tumour lies between the heart and the thoracic wall, there may be an intensification of the cardiac sounds. It may happen that pericarditis assumes the characters of gastric irritation or inflammation.There will be nausea.The recognition of the affection will result from an examination of the region of the heart.

Endocarditis of a simple and acute character is very commonly encountered in rheumatic fever cases,and usually during the second week of the disease.Sometimes

it precedes the articular manifestation of the rheumatic affection.In estimating the etiological importance that rheumatic fever bears in the production of endocarditis it must be remembered that not every blowing sound or murmur is indicative of an inflamed endocardium. In most of the cases the occurrence of simple acute endocarditis is not expressed by any special symptoms in connection with the heart.Its advent is so insidious,and its symptoms and physical signs are so uncertain,that in its early stages one always hesitates to state positivelythat it exists.The symptoms which excite suspicion are a sense of distress,sometimes amounting to pain, in the praecordial region.In children the pain in often referred to the epigastrium and is accompanied by palpitation.The pulse becomes feeble and sometimes dicrotic.The patient is restless and anxious and prefers a half-sitting posture.A hacking cough may be present,accompanied by slight dyspnoea.Variations in temperature are not constant or significant.A slight rise in temperature,however,is often the only symptom of an acute endocarditis complicating chronic valvular disease,and when the rise of temperature in such a case persists for a number of days without an assignable cause,it is a strong diagnostic point.The duration of simple acute endocarditis varies from a fortnight to six weeks.In most of the cases convalescence is established in a month.In the early stages inspection and palpation may show an increase in the force and rapidity of the cardiac impulse;later the force of the impulse is diminished.There is nothing,however,diagnostic either in these changes or in the changes in the area of praecordial dulness.The most significant,but by no means certain, physical signs of simple acute endocarditis are adventitious sounds or murmurs which replace the normal heart sounds.Blowing sounds accompanying or taking the place of the heart sounds which are not due to endocarditis, but to the attendant blood changes and muscular incompetency,are,however,so frequently heard in acute rheumatism that the occurrence of a murmur cannot be regarded as evidence of endocarditis.If,however,during the first two weeks of acute rheumatism a murmur is developed which has been preceded for some time by a prolongations of the first sound of the heart,the probability is that the endocardium is affected.Usually under such circumstances there will be other evidences of disturbance of the circulation,such as palpitation accompanied by distinct interference with the force and rhythm of the heart's action.It must be borne in mind that a murmur alone is very unreliable evidence of the occurrence of endocardial inflammation.One sometimes notices,two or three months after recovery from an attack of rheumatic fever in vigorous adults,that murmurs and cardiac symptoms develop,which leave no doubt that an endocarditis has accompanied the rheumatic attack,though during its acute stage it had not been attended by a murmur.Simple acute endocarditis is in rare instances complicated by embolism and the development of infarctions.When pleuropneumonia complicates this variety of endocarditis it is usually of embolic origin.While the diagnosis in a large proportion of cases of simple acute endocarditis must always be uncertain,in a number of instances where the evidences of the existence of endocarditis are well established it is difficult to determine whether it is

simple or malignant.Usually the history of the case and the renal or visceral or other complications are sufficient to indicate a malignant endocarditis.There are, however,cases of malignant endocarditis which are so mild in type that one does not suspect their malignant nature until an unexpected fatal issue reveals their true nature.

Malignant endocarditis sometimes occurs in connection with rheumatic fever,though the frequency of this has certainly been overestimated.This may be in part due to confusion of the simple with the malignant form,and in part to the fact that the pain in the joints may be called rheumatism,although in reality only a symptom of the infectious process with which the endocarditis is associated.On the other hand,the painful affection of the joints which may appear after the endocarditis is established is frequently septic in nature,though it may be rheumatic.The lesions vary in different cases, being either vegetative,ulcerative,or suppurative.The vegetations of the malignant form are generally much more pronounced than in the simple form,though otherwise similar.They consist of irregular,often cauliflower-like,masses of granular fibrin,entangling desquamated endothelial cells,round cells,and microbes.In some cases by detaching the vegetation the base is found to be distinctly ulcerated;in other cases it is only slightly roughened and elevated.The vegetations themselves may be quite tough;more commonly they are friable and easily detached.In the cases to which the term ulcerative is especially applicable,and which form a large proportion of the whole number,distinct ulcers are found upon the valves or mural endocardium,sometimes shallow, more commonly deep and destructive.Occasionally the ulcer is so covered by fibrinous deposit as to escape detection until the vegetations are removed,and in a few cases spots of superficial erosion or roughening may be found before the actual ulceration has occurred.In cases of suppurative type small purulent collections between the valvular reduplications or at the base of the valves,and involving the walls of the cavities,are seen.These small abscesses may remain intact and be found at autopsy;or they may break and thus produce superficial ulcers.The situation of the lesions is practically the same as in simple endocarditis,the mitral and aortic valves being affected much oftener than the tricuspid and pulmonary.In the two hundred and four cases observed by Osler the mitral valve was alone affected in seventy-seven,the aortic in fifty-three,the tricuspid in five,and the pulmonary in four.Coincidently with disease of the valves or independently the mural endocardium is involved in a considerable number of cases,the points of greatest frequency being the upper part of the septum ventriculorum and the posterior wall of the left auricle.Very often areas adjoining the ulcerations or vegetations are secondarily affected by contact during the cardiac movements,and in rare instances the lesions may spread to the lining of the aorta. The local results of the ulcerative lesions are mainly of a destructive nature.The valves may be so eroded that they are reduced to mere stumps;the leaflets or the septum may be perforated;one side only of a leaflet may be destroyed and an acute valvular aneurism result; or,in case of involvement of the mural endocardium,

even partial cardiac aneurism may occasionally occur. The formation of cardiac or valvular aneurism, however, is a rare occurrence, and still less commonly aortic aneurisms have been described at the seat of endarterial ulcers. Purulent myocarditis or even pericarditis may result from direct extension, but the former is more often due to miliary embolism of the coronary arteries, and the latter generally arises coincidently with endocarditis rather than secondarily. The remote lesions of malignant endocarditis are the outcome of septic intoxication or of embolism. The spleen is generally enlarged as in infectious diseases, and the parenchyma of the kidneys and liver suffers the degenerative changes of fever. To these lesions may be added those of embolism. In case of mitral or aortic disease this involves the systemic circulation, and the emboli are lodged in the spleen, the kidneys, the brain, the cutaneous vessels, or the retina; in case of tricuspid or pulmonary valvulitis the lungs are the seat of embolic lesions. In many cases simple haemorrhagic infarcts result, but in the more decidedly pyaemic cases suppurative infarctions or multiple miliary abscesses are observed; and sometimes a central point of suppuration is surrounded by a more extensive zone of haemorrhage. In the skin, the serous surfaces, and the retina minute haemorrhages may result from embolism or from degenerative changes in the vessels unassociated with emboli. Cerebral embolism leads to lesions of the meninges or of the deeper structures. There may further be meningeal or deeper haemorrhages, or embolic softening, simple or suppurative in nature. In some instances multiple pyaemic deposits have been found in the brain. Pleurisy, parotitis, and embolism with secondary ulcerations in the stomach and intestines are among the rarer lesions. When the spleen is the seat of infarctions, a moderate degree of local peritonitis may be seen. The symptomatology of malignant eneocarditis presents such variety in different cases that no description will closely apply to all of them. In nearly all instances, however, there is an underlying, if not conspicuous, pyaemic element, which gives to the disease its most marked peculiarities. The onset is usually abrupt and marked by a decided chill. In the case of the rheumatic patient a considerable exacerbation of the temperature occurs. During the continuance of the disease irregular remittent or sometimes definitely intermittent fever is maintained, the evening temperatures being 103. to 104. F. in decided cases, and the morning a few degrees or even subnormal. With this irregular fever repeated rigors and drenching sweats may be associated, and may add to the depression of the general strength of the patient, which in any case is a marked symptom. Local indications may or may not be present. Generally there is slight oppression or the feeling of constriction at the heart; more rarely there is actual pain, but severe pain is very unusual. The cardiac action is very excited, the pulse is rapid, and often irregular and weak. A slight subjective sense of palpitation may be the only symptom during the course of the disease to draw attention to the heart. Dyspnoea is less decided than the rapidity of the pulse would lead one to suspect, unless the affection is complicated by lesions in connection with the lungs. The tongue is coated, and in many cases becomes brown and dry. Sordes collect about the teeth. The stomach is frequently irritable in the early stages, and

vomiting may prove an urgent symptom.The abdomen is often distended with gas,and diarrhoea may alternate with constipation,or may be so persistent and severe as to resemble cholera.The spleen is found enlarged and tender.The urine nearly always contains albumin,and may even be tinged with blood.Tube-casts,epithelial and granular,are frequently found.Nervous symptoms are seldom absent.They may merely consist of headache,restlessness,or slight muttering at night,but in severe cases,and especially towards the close,active delirium and a soporose or completely stuporose condition may be developed;and when cerebral embolism has supervened the occurrence of various palsies may serve to indicate the nature of the cerebral complication.The entire aspect of the disease may be wholly changed by the supervention of embolic manifestations.Sharp pain or tenderness or swelling of the spleen denote infarction and localised peritonitis about this organ.Small emboli of the kidney may not declare themselves by any definite symptoms.In the case of larger infarcts,however,pain in the lumbar region and haematuria are distinctive symptoms.Albuminuria alone occurs in nearly all cases,and is due to parenchymatous degeneration of the kidney,but when emboli obstruct the renal vessels the quantity of albumin increases notably.The manifestations of cerebral embolism depend upon the seat and resulting lesions. Consciousness is nearly always lost,the patient becoming stuporose or comatose,and palsies result from occlusion of the vessels supplying the motor cortex.Dimness of vision or extensive inflammation of the eye may follow retinal embolism,and colicky pain,gastric disturbances,and diarrhoea,sometimes haemorrhagic in nature,give evidence of the occlusion of the gastric or intestinal vessels.Icterus is an occasional symptom,and may be due to the septic and febrile degeneration of the liver or to embolism of one of the branches of the hepatic artery,as has been seen in a few instances.The skin may also present a vivid-red erythematous eruption, especially in the early stages,and later petechiae are not uncommon.The latter may be due to minute emboli or to degeneration of the vessels without occlusion.They are most numerous upon the abdomen and chest,and sometimes have a distinctly papular as well as petechial character.In a large number of cases the disease resembles closely typhoid fever in its symptomatic indications,and it is customary to speak of this as the typhoid form.In certain cases,in which the onset is not abrupt,but gradual and attended by malaise or other prodromal symptoms,the resemblance to typhoid fever may be most deceptive.Later nervous and abdominal symptoms become pronounced,the patient sinking into a delirious or stuporous condition,picking at the bedclothes,and slipping down to the foot of the bed as in typhoid fever.The tongue becomes dry and brown,sordes collect around the teeth and gums,the abdomen is distended,the spleen enlarged,the bowels are apt to be relaxed,and tenderness in the right iliac fossa may be present from the first.To this may be added the eruption,which,though not like the characteristic rash of typhoid fever,is simulated in certain cases of the latter disease. The temperature is high and usually remittent,but at times may be continuous at 103.or 104.F.during the entire course of the disease.In another group of cases the

affection is manifestly of pyaemic nature, and to such the term arterial pyaemia, applied by Wilks, is not inappropriate. It is this type that occurs most frequently in the severest forms of rheumatic fever under septicaemic suppurative conditions. The onset is abrupt and attended by a decided chill. Throughout the disease the temperature is highly remittent or even intermittent, and attended by more or less periodical chills and sweats. The patient becomes prostrated, the tongue is brown, the breath offensive, the skin sallow or jaundiced, and emaciation may proceed rapidly to an extreme degree. Embolic processes of a suppurative nature and a petechial eruption may finally call attention to the cardiac lesion. There sometimes occur cases in which, with irregular fever of remittent type, copious sweats, and copious sweats, and distinct endocarditis, articular symptoms appear. Various joints may be affected in a more or less fugitive manner, or one or two of the larger joints may be distinctly inflamed. It may be difficult for some time to decide that the fever is not rheumatic and the endocarditis and arthritis dependent upon it. The malignant, like the simple, form of endocarditis may present few signs in certain cases. The most frequent physical sign is the systolic blowing murmur heard in the region of the apex, and generally indicative of diseaseof the mitral valve. It must be remembered, however, that systolic murmurs, not at all dependent upon endocarditis, are often heard during the course of fevers. An aortic systolic, or more rarely diastolic, murmur may be present, and the latter especially is significant of valvular disease. In all cases it will be found that the sounds alter from day to day in character and position of maximum intensity. Inspection and palpation merely reveal the overaction of the heart, and in protracted cases percussion may show a slight enlargement, but in ordinary cases this is rarely found. The existence of previous chronic valvular disease would, of course, be indicated by the altered character of the physical signs, in accordance with the particular lesions present. The most important complications are those which result from embolism; they have been sufficiently detailed already. Pericarditis and myocarditis from extension or from embolism are conditions of great gravity, and manifest themselves by the unusual and early depression of the heart's action and by physical signs varying with the exact nature of the lesions. Pneumonia and pleurisy are occasionally met with, though the former is more frequently antecedent to the endocardial lesion. The pleural effusion is not rarely purulent in character. Gastric and intestinal derangements may sometimes reach a severe grade, even without embolism, and vomiting and excessive diarrhoea, almost choleraic at times, may continue throughout the case. Meningitis is most commonly met with in the malignant endocarditis following pneumonia. It may be an early complication, and its symptoms may completely dominate the attack. The diagnosis of malignant endocarditis is often extremely difficult from the fact that the cardiac condition is sometimes latent or overshadowed by severe complications. The disease is most frequently mistaken for typhoid fever, rheumatic fever, or ordinary pyaemia. The distinction from typhoid fever is especially difficult when the endocarditis is obscure in origin and when prodromal malaise is noted. As a rule, however,

the onset of malignant endocarditis is abrupt, the temperature is irregular, there are more or less well-defined chills and sweats, and there may be great rapidity of the pulse, oppression, or pain to direct attention to the heart. The abdominal distention and splenic enlargement of typhoid fever are usually more decided than in malignant endocarditis, and the eruptions are essentially different in typical cases. The frequency with which precedent organic cardiac disease will be discovered is an important point, as is also the much greater frequency of albuminuria with casts in malignant endocaridit is. Other simulating conditions must be distinguished on general argumentative lines and according to the history and general phenomena of the case.

Chronic valvular disease is a very common outcome of a rheumatic attack. Aortic stenosis gives rise to a greater or less narrowing of the orifice of that vessel, and also hypertrophy of the walls of the left ventricle; some insufficiency usually accompanies the lesion. There are no subjective symptoms so long as compensation is complete. Even quite extensive stenosis is not incompatible with a condition of perfect health, for a perfect compensatory hypertrophy enables the heart to fill the arterial system and relieve the pulmonary pressure. When the ventricular hypertrophy no longer compensates for the obstruction, the arteries are filled inadequately at each systole, the left auricle cannot empty itself freely into the left ventricle, and the pulmonary vessels, as well as the entire venous system, become abnormally full. The diminished arterial supply, gives that pallor to the face which so frequently accompanies this condition, and syncope is liable to occur as a result of cerebral anaemia. These are late effects, and do not usually appear until a condition of more or less mitral insufficiency is reached. As a rule, signs of arterial anaemia, such as pallor, cold hands and feet, loss of muscular power, sense of langour on slight exertion, impaired nutrition, giddiness, nausea, and all the other signs of central anaemia, precede the evidence of venous engorgement. With the venous congestion there are the usual symptoms of engorgement of the pulmonary and systemic circulations. The pulse in aortic stenosis is normal in frequency, but diminished in volume and force. Usually it is regular in rhythm, although it may be intermittent and jerky in character. The sphygmographic tracings show a slanting or oblique up-stroke and considerable separation between the percussion and the tidal waves. Cerebral embolism is associated with aortic stenosis more frequently than with any other valvular lesions; the embolus usually lodges in the left middle cerebral artery. Sometimes embolism is due to small auricular or ventricular clots that form behind the obstruction. Such clots occlude the aortic orifice and cause sudden death. Usually the physical signs of aortic stenosis are distinctive and easily appreciated. In most cases the visible area of the cardiac impulse is seen on inspection to be abnormally increased, and the apex is displaced downwards and outwards, frequently accompanied by a lifting of the chest wall over the heart. In old subjects with rigid chests and emphysematous lungs, even when there is extensive hypertrophy, there may be no visible impulse. On palpation, if the chest walls are rigid, the apex beat may be imperceptible; if they are

yielding,it is usually heaving and forcible in character.A systolic thrill of great intensity is often felt at the base of the heart,having its point of maximum force in the second right intercostal space.On percussion,the increase in the area of cardiac dulness will correspond to the displacement of the apex to the left,and measures the degree of hypertrophy of the left ventricle. On auscultation it will be found that aortic stenosis produces a systolic murmur which more frequently accompanies than replaces the first sound of the heart. The maximum intensity of this murmur is usually at the second sterno-costal articulation of the right side.It may be heard,however,with equal intensity over the whole of the upper portion of the sternum,and may be transmitted up the aorta and into the carotids.It sometimes has its maximum intensity at the junction of the third left rib with the sternum.In most cases the first sound of the heart is heard with the murmur,but the latter may entirely replace or obscure it.This murmur is loud and harsh in character,and is loudest at the beginning of the systole.In pure aortic stenosis the aortic second sound may be inaudible,owing to the thickening and rigidity of the aortic valves,but the pulmonic second sound is always very distinct.The area of diffusion of this murmur follows the law that a murmur is propagated in the direction of the blood-current.Sometimes it may be heard in the thoracic or abdominal branches of the aorta.It mu t be remembered,however,that a systolic murmur,audible at the base and carried along the ascending arch towards the sternal end of the right clavicle,is not limited to cases of aortic stenosis,although aortic stenosis always produces a murmur with these characteristics.The diagnosis is usually established by the discovery of a rough systolic murmur,with its maximum intensity at the second right costal articulation, transmitted along the ascending arch to the carotids, accompanied by a thrill,a hard,slow pulse,and the evidence of left cardiac hypertrophy in one past middle life.If one,however,rests entirely upon these signs,he is liable to mistakes,for a roughened and calcified aortic valve with an atheromatous aorta in one who has cardiac hypertrophy due to chronic nephritis may present almost the same signs.It is to be remembered also that systolic murmurs heard in this region may be produced by mitral and tricuspid insufficiency and by anaemia, but the point of maximum intensity of the murmur and the direction of its transmission will readily distinguish these murmurs from that of aortic stenosis,- which latter is hear with its greatest intensity at the second right sterno-costal articulation,and diminishes in intensity towards the apex.The murmur of mitral insufficiency is heard with its greatest intensity at the apex.The murmur of aortic obstruction is transmitted into the vessels of the neck;that of mitral regurgitation to the left,toward the apex beat,and is heard behind,between the fifth and eighth dorsal vertebrae,to the left of the spine,with almost the same intensity as at the apex.Tricuspid insufficiency also is accompanied by a systolic murmur,but it is rarely audible above the third rib,and its area of diffusion is seldom more than two inches from the point of its maximum intensity at the junction of the left fourth rib with the sternum. An anaemic murmur is systolic,and heard over the upper

portion of the sternum and transmitted into the vessels of the neck, but its maximum intensity is over the carotids. It is soft and blowing in character, and is accompanied by the subjective signs of anaemia and the characteristic venous hum. Thoracic aneurisms may produce murmurs resembling those of aortic stenosis, but the dilating impulse, the single or double bruit, the pain, and the usual symptoms of thoracic aneurism are absent when aortic stenosis alone is present. Furthermore, aneurismal murmurs have their maximum intensity at the seat of the aneurism, and not at the base of the heart.

Aortic insufficiency, so long as there is full compensatory hypertrophy of the walls of the left ventricle, gives the patient little or no inconvenience, even when the lesion is considerable. The uncomplicated affection often exists for years without giving rise to any sensations of distress about the heart that will attract the attention of the patient or in any way interfering with his moderately active existence. In those cases in which the aortic insufficiency is associated with it is part of a general arterio-sclerosis the failure in compensation occurs earlier than in those in whom the arteries are comparatively normal. As soon as there is the slightest failure in compensation the cardiac action becomes excessive during excitement or violent muscular effort, which causes the individual to become anxious and irritable, and he is generally aware that exercise will augment his uncomfortable symptoms. As the hypertrophy increases he suffers from dyspnoea, cardiac palpitation, vertigo, flashes of light, a feeling of faintness on rising suddenly, and is forced to sleep with his head raised. On slight exertion the patient experiences a sense of cardiac oppression and palpitation. There is a visible pulsation in the arteries of the head, neck, and upper extremities. Pain in the precordial region, in the left shoulder, or extending down the left arm becomes a troublesome symptom, and is usually of a distinctly shooting or stabbing character. Sometimes this pain is accompanied by numbness and a peculiar whiteness of the skin along the line of pain. In certain cases the pain shoots from the middle of the sternum to the right arm. As the failure in compensation increases, attacks of dyspnoea occur at night, and the patient is often unable to lie down on account of the difficulty in breathing which comes on as soon as he assumes the recumbent posture. The feet become oedematous, and in some instances the oedema gradually extends upwards until a condition of general anasarca is reached. The face becomes puffy and cyanotic. In advanced cases there is orthopnoea and angina pectoris. Attacks of syncope at first occur only on exertion, but later they come on independently of it, and are very distressing. The pulse is characteristic. It was first described by Sir Dominick Corrigan, and is usually called the Corrigan pulse; it is sometimes also spoken of as the piston pulse. It is large and distinct, quickly projected against the finger, and just as quickly the arterial tension sinks and the impulse vanishes. Sometimes it is accompanied by a vibrating jar. Its characteristics are more apparent when the arm is raised above the head. Although quick and jerking, it is always regular in rhythm. The radial pulse is felt a little after the apex beat; thus the pulse wave of aortic regurgitation travels along the arteries.

The delay in the pulse is always constant.Pulsation of the retinal arteries can often be detected by the use of the ophthalmoscope.In advanced cases the patient becomes irritable and peevish.Sometimes delirium with hallucinations and suicidal tendencies develop.He may have to be forcibly restrained from killing himself or his attendants.Inspection of the chest shows a forcible and increased area of cardiac impulse,sometimes reaching to the seventh interspace and laterally to the left axillary line.The vessels of the neck are seen to pulsate forcibly,and at times all the superficial vessels have a distinct throbbing impulse.The so-called capillary pulse,although sometimes seen in this condition,is not constantly or necessarily connected with it.On palpation a heaving,lifting impulse will usually be perceived over the praecordial region,which may be transmitted over a large portion of the anterior chest wall. When there is extensive dilatation of the left ventricle the impulse will become diffused and indistinct.Occasionally a continuous diastolic thrill,equally intense during the whole of the diastole,will be felt over the sternum,most distinctly at the site of the aortic valves.The area of percussion dulness corresponds to the degree of cardiac involvement.Deep dulness is elicited below and to the left of the normal cardiac area,and its outline is more or less oval than in health.So soon as dilatation exceeds the hypertrophy the area of dulness will extend laterally rather than vertically.The area of dulness may extend six to eight inches from right to left and from the upper edge of the third rib to the line of the liver dulness.The superficial area of dulness may be increased laterally and towards the left.The affection is seen on auscultation to be characterised by a diastolic murmur which may take the place of,or immediately follow,the second sound of the heart.It is distinct at any point over the base of the heart,but usually has its maximum intensity either at the sternal end of the second right costal cartilage in the second right intercostal space, or at the sternal junction of the third rib on the left side.Itis transmitted over the sternum,and sometimes will be loudest at the xiphoid cartilage.Sometimes it is transmitted towards the apex.Its area of diffusion is greater than that of any other murmur.It is not only conducted down the sternum to the xiphoid cartilage and to the apex,but sometimes it may be heard faintly in the transverse and descending portions of the arch,along the spinal column,and even in the radial and femoral arteries.Foster says that the incompetency of the posterior segment of the valve produces a murmur which is conducted to the apex,whereas inadequacy of either or both of the anterior segments is accompanied by a murmur which is conducted to the ensiform cartilage.This point has a practical bearing on account of the relation of the anterior segments of the valve to the coronary artery.If the murmur indicates a lesion of the posterior flap of the valve,the prognosis is better. The murmur of aortic incompetency may be soft,blowing, or rough,and is frequently musical in character.It is loudest at the beginning of diastole and gradually decreases in intensity.If aortic stenosis coexists with aortic incompetency,there will be a double murmur, having its maximum intensity at the right side of the sternum in the second intercostal space.The systolic

and diastolic portions of such murmurs are sometimes separated from each other by a distinct pause.If mitral incompetency is associated with aortic incompetency, each murmur will retain its own area of maximum intensity and diffusion.Murmurs of aortic incompetency are sometimes very indistinct,and can only be heard when the patient is in the recumbent position.The loudness of the murmur is not a measure of the extent of the incompetency.A diastolic murmur heard at or below the line of the aortic valves,chiefly aurdible in the centre of the sternum,indicates extensive aortic incompetency.If a diastolic murmur is audible in the carotids, it is invariably preceded by a loud systolic murmur.

Patients suffering from mitral stenosis present few subjective symptoms.Active physical exertion causes cardiac palpitation,but this will cease as soon as the auricle can relieve itself,which is readily accomplished by the patient's lying on the right side with the head slightly elevated.These patient are usually pale and anaemic.Anginal pains are frequently felt in the region of the apex.As long as compensation is maintiained by the auricular hypertrophy the pulse is regular and normal in character.When the left ventricle no longer receives and discharges the normal quantity of blood,the pulse becomes small,feeble,rapid,and irregular.The pulmonary congestion which attends the advanced stage of mitral stenosis causes continual dyspnoea, which is increased by physical exertion and is accompanied by a hacking cough.After prolonged or violent physical exertion a large quantity of glairy,watery mucus may be expectorated in a few months or a profuse watery,blood-stained expectoration indicative of pulmonary congestion and oedemamay occur.The exertion of walking rapidly against a strong wind has been known to induce such intense congestion and oedema of the lungs in patients with mitral stenosis as to cause death suddenly.Orthopnoea is not a frequent symptom of mitral stenosis,for even in extensive and long-standing cases the pulmonary circulation is maintained,since the auricle is usually able to empty itself,and only becomes engorged during active physical exercise.It is erroneous to suppose,as formerly,that pure mitral stenosis produces hypertrophy of the left ventricle.On inspection the impulse of the heart is usually seen to be feeble and indistinct.Sometimes it has a visible undulating movement.It is seldom perceptible to the left of its normal area,but is usually most distinct in the fourth left interspace near the sternum.On palpation the apex beat is less forcible than usual.A distinct presystolic, purring thrill will be communicated to the hand. This thrill is the diagnostic sign of mitral stenosis.It is most distinct in the region of the apex.It begins during the diastole,and increases in intensity up to the commencement of the ventricular systole.It is often perceptible as high as the third interspace,and sometimes as high as the second.It may be diffused over the whole praecordial area and may continue throughout the entire systole of the heart.On percussion the area of cardiac dulness will be increased to the right and upwards.If the left auricle is greatly dilated,the increase in the dulness will be upward along the left border of the sternum.Mitral stenosis is characterised by a loud churning,blubbering presystolic murmur.This

murmur is longer in duration than any other murmur.It ends at the commencement of the first sound, being synchronous with the purring thrill.It is heard with its maximum intensity a little above the apex beat.It is always louder when the patient is erect than when in the recumbent posture.It is seldom conveyed to the left of the apex beat,and loses in intensity as the stethescope passes to the right of the sternum or above the third rib.The second sound of the heart in the second left interspace is loudly accentuated and sometimes reduplicated.If the murmur immediately follows the second sound and continues through to the commencement of the first, it indicates a diaphragmatic construction of the mitral orifice.When mitral stenosis and incompetency coexist the two murmurs run into each other,constituting a single murmur.The harsh character of the presystolic element of the murmur can always be recognised.As compensation fails a stenotic murmur loses its intensity and often becomes inaudible;if the compensation is restored,the murmur reappears.The occurrence of the stenotic affection may pass unnoticed if the patient is seen during the period of broken compensation.

In mitral incompetency there are no subjective symptoms during the early stage,unless the condition comes on suddenly from rupture of the valve or of the chordae tendineae;and so long as hypertrophy of the right ventricle perfectly compensates for the insufficiency,even though the hypertrophy is extreme,the patient will not be made aware of its existence.But when the right ventricle fails to overcome the obstruction in the pulmonary circulation caused by the regurgitant blood-current,and the stage of commencing failure of compensation is reached,there will be dyspnoea on exertion,accompanied by cyanosis and a hacking cough,with expectoration of frothy serum.Sometimes the latter is blood-stained,and there may be attacks of quite free haemoptysis,although it should be remembered that haemoptysis is far more frequent with mitral stenosis than with incompetency.This stage is often marked by palpitation of the heart.In the advanced stage,when the period of broken compensation is reached,the lips,face,and finger-tips become blue and the symptoms of extensive venous engorgement are present.The heart action becomes weak and irregular,dyspnoea and cough are constant,and the watery-blood-stained expectoration contains brown pigment granules.The liver is enlarged,and the patient will complain of a sense of weight and fulness in the hypochondrium,and there will be anorexia,nausea,and a sense of oppression in the epigastrium.Sometimes the hepatic circulation becomes so obstructed that jaundice will be added to the cyanotic discoloration,which gives to the surface a peculiar greenish hue.Congestion of the portal circulation causes frequent attacks of gastric and intestinal catarrh,and evidences of embarrassment of the renal circulation are present.The urine is highly coloured and loaded with urates,and may contain albumin and blood casts.Headache,dizziness,vertigo,stupour,somnolence,and sometimes a peculiar form of delirium often occur.A late symptom of mitral incompetency is dropsy,beginning in the feet and gradually extending to the trunk and serous cavities.It may require several years or only a few months before a condition of general anasarca is reached.With the anasarca the dyspnoea

becomes extreme, the patient being unable to lie down at a time for more than a few moments. In this stage the occurrence of haemorrhagic infarctions in the lungs is not infrequent. As these symptoms come on gradually, and as the compensation may be temporarily restored by treatment, these patients are apt to become very hopeful and to expect much from their medical adviser, but ultimately they reach a condition for which no relief can be given, and they die either from general dropsies or excessive dilatation of the heart. The fatality is of slow occurrence. The pulse of mitral incompetency before the period of failing compensation remains regular in force and rhythm; after the stage of failing compensation has been reached it becomes diminished in force and volume, irregular in rhythm, and increased in frequency, but never jerking in character. When the heart's action is excited it has a certain tremulousness. These last-mentioned characteristics are to be regarded more as a result of the changes in the myocardium than of the valvular incompetency. If the pulse of mitral insufficiency has any distinctive characteristic, it is its diminution in volume. On inspection the visible area of cardiac impulse is abnormally increased and is more or less distinct, according to the extent of the right ventricular hypertrophy. Sometimes in children there will be bulging of the praecordium and a heaving of the thoracic wall with each systole, and not infrequently there is an epigastric impulse synchronous with the heart beats. In aggravated cases a double impulse often accompanies the cardiac systole, and is due to a non-coincidence in theconctraction of the ventricles. The jugular veins may be distended and have a wavy impulse when the patient is in a recumbent posture. The apex beat on palpation is found to be displaced to the left. If the hypertrophy predominates over the dilatation, it is felt below the normal area; when the dilatation exceeds the hypertrophy, the apex beat is carried outwards and slightly upwards. The impulse is diffused and more or less forcible according as the right or left ventricular hypertrophy predominates. A systolic tremor, felt most distinctly at the apex and becoming less intense the farther the hand is removed either to the right or left from that point, is invariably due to mitral insufficiency. It is seldom, or never, that one meets with a distinct purring thrill in mitral incompetency unassociated with mitral stenosis. The area of superficial as well as deep dulness is increased laterally and downwards on percussion. There is no valvular disease which produces such extensive lateral increase in the area of dulness as mitral incompetency. On auscultation it will be found that mitral incompetency causes a systolic murmur which either completely or partially replaces the first sound of the heart. The quality of the murmur is variable, and not in itself as distinctive as that of mitral stenosis. It is usually soft and blowing in character. Sometimes towards its end the murmur becomes distinctly musical in character. It is heard with its maximum intensity at the apex, and its area of diffusion is to the left. It can be heard at or at the inferior angle of the left scapula, and is usually as distinct between the lower border of the fifth and the upper border of the eighth vertebra at the left of the spine as at the apex. It varies in intensity withthe position of the patient. It may be present in the

recumbent and absent in the erect posture.Accentuation of the pulmonary second sound is an important sign in mitral incompetency,and is heard with maximum intensity at the second interspace to the left of the sternum.Skoda first drew attention to this sign,and regarded it as an infallible indication of the lesion we are now considering.When mitral incompetency and stenosis coexist, a continuous murmur is heard,which begins usually after rhe second cardiac sound and continues throughout the cycle of the heart.The two murmurs,although mingling to form one,can in most instances be readily distinguished from each other,for the point of maximum intensity and the very limited area of diffusion of a presystolic murmur readily distinguish it from a mitral systolic.It is important to recognise the existence of both these murmurs in forecasting a case.The diagnosis should be easily effected in mitral incompetency.The rhythm and area of diffusion of the murmur which attends it are sufficient to distinguish it from other valvular lesions.The character of the pulse,the symptoms referable to the right heart,and the pulmonary complications will also assist in its diagnosis.Sometimes systolic murmurs are produced in the left ventricle which simulate very closely the murmur of mitral insufficiency,but such murmurs are not associated with ventricular hypertrophy or accentuation of the pulmonary second sound.It is not possible to determine in every instance by its character whether a murmur of mitral insufficiency is due to a lesion of the valve segment or to relative mitral insufficiency from dilatation of the mitral ring;but the recognition of the affection will be assisted by the condition of the arteries and the presence of insufficiency of the aortic valve.

Pulmonary stenosis has no constant nor diagnostic subjective symptoms.In some of the published cases there were murmurs,in others there were cardiac palpitation,dyspnoea,cyanosis,and anasarca - none of which belong to the pulmonary lesion or necessarily depend upon it.Inspection,palpation,and percussion give negative rather than positive results.In a few cases an appreciable thrill may be felt,confined to the region of the second left intercostal space near the sternum. On auscultation a systolic murmur may be heard with its maximum intensity immediately over the pulmonic valves. It is very superficial,very distinct,and limited in its diffusion.It is not transmitted to the xiphoid cartilage not along the course of the aorta nor into the vessels of the neck.The pulmonary second sound is weak or absent.It may be obscured by a diastolic murmur. Some of the cases present hypertrophy of the right ventricle.The lesion can only be diagnosed by a process of exclusion.It may be differentiated from aortic stenosis by the absence of the characteristic pulse of the latter,the absence of left ventricular hypertrophy,and the fact that the murmur is not transmitted into the vessels of the neck.It is impossible to distinguish a murmur produced by pulmonic stenosis from one produced by pressure on the pulmonary artery,by an aneurism at the sinus of Valsalva,or a tumour in the anterior mediastimum,nor can it be distinguished from the soft blowing murmurs sometimes expressive of the pressure exerted by a consolidation of tuberculous origin.

The symptoms of pulmonary incompetency are not

distinctive.A diastolic murmur may be heard with greatest distinctiness in the second right intercostal space, and is transmitted to the lower part of the sternum.It is difficult to distinguish this murmur from that of aortic regurgitation,though the enlargement of the right ventricle,the establishment of tricuspid insufficiency and early cyanosis,and the absence of the water-hammer pulse would furnish strong indications of pulmonic disease.Furthermore,it may be noted that the pulmonary murmur is loudest to the left of the sternum,the aortic to the right of the latter.

The physical signs of tricuspid stenosis are seldom definite or decided.Inspection shows a jugular pulsation and an epigastric impulse.A venous thrill will usually be felt at the base of the neck.The area of cardiac dulness will be abnormally increased laterally and upwards to the right.On auscultation of presystolic murmur is heard,with its maximum intensity at the lower portion of the sternum just above the xiphoid cartilage. This murmur may be transmitted faintly towards the base of the heart,but never towards the apex.The diagnosis of this lesion can only be made by exclusion.The murmur of mitral stenosis,with which tricuspid stenosis is almost always associated,is limited to the apex;the murmur of tricuspid stenosis,having the same rhythm,is heard at the base of the ensiform cartilage,and between these points there is a region without any murmur at all.

Incompetency of the tricuspid valve being usually secondary to some mitral lesion,its symptoms will during its early stage be vague and masked by those of the primary affection.But as soon as the valves become so inefficient that the venous return is markedly impeded, a train of symptoms is developed which have their origin in the visceral changes already referred to.With extensive tricuspid insufficiency there may be cardiac palpitation,dyspnoea,and marked irregularity in the force and rhythm of the heart.The hepatic and splenic areas of dulness are increased,the skin becomes shiny,and there is obstinate constipation.Dyspeptic symptoms are prominent and the urine is scanty,dark in colour,and of high specific gravity.Marked cerebral hyperaemia is manifested by headache,dizziness,and vertigo.There is a peculiar mental disturbance which is not met with in any other form of heart disease.If the patient is placed in a recumbent position,the face becomes turgid and blue,and if he remains long in such a position,stupour and coma may supervene.A very late symptom is dropsy, which begins at the ankles and extends upwards until a condition of general anasarca supervenes,the genital organs rarely becoming oedematous.On inspection the visible area of the cardiac impulse is increased more in extensive tricuspid insufficiency than in any other valvular lesion.It sometimes extends from the left nipple to the xiphoid cartilage.There is an impulse in the jugular veins,more apparent in the right than in the left.Pulsation may be observed in the veins of the face,arms,and hands.On palpation the apex beat is diffused and feeble except when the left ventricle is greatly hypertrophied.There is distinct epigastric pulsation,due to reflux into the veins of the liver, which is synchronous with the cardiac impulse.Percussion will reveal an increase in the area of cardiac dulness upwards and to the right,sometimes as high as

the second intercostal space.The murmur heard on auscultation in these cases occurs with,or takes the place of,the first sound of the heart.It is superficial,low-pitched,faint,soft,and blowing,and is heard with greatest intensity over the lower part of the sternum.As a rule,it is inaudible above the third rib or to the left of the apex beat.Sometimes it is transmitted from the base of the xiphoid cartilage two or three inches upwards to the right of the sternum.It may be overlooked when it is audible only over a limited area.The murmur of tricuspid insufficiency must not confounded with the murmurs of aortic and pulmonary obstruction.It must be borne in mind that a tricuspid regurgitant murmur is never audible above the third rib,is not attended by accentuation of the pulmonic second sound,and is accompanied by jugular and epigastric pulsation.Its point of maximum intensity is near the base of the ensiform cartilage.It is not difficult to differentiate it from a mitral regurgitant murmur if the rules for the diagnosis of such a murmur are kept carefully in mind.

Acute dilatation of the heart,we have seen,may occur in rheumatic fever cases.Breathlessness occurs suddenly and the chest feels as if bound down by a constricting band.A sense of oppression is felt over the praecordial or epigastric region.Dizziness is marked,and nausea may or may not be present.The action of the heart is rapid,the pulse is small,and,it may be,irregular or intermittent.The skin,particularly of the face,assumes an ashy-gray or leaden hue,while the lips are of a dark-purple colour.The vision becomes obscured,and flashes of light pass before the eyes.The ideas become confused, the intellect clouded,and the patient perhaps lapses into unconsciousness.All these symptoms may occur without actual dilatation of the heart,at least of a permanent nature,and after a few hours or a few days the patient returns to normal condition.Even in these cases, however,cardiac palpitation and dyspnoea are likely to come on after some time when the patient exerts himself. On the other hand dilatation may occur from which the individual recovers only after months or years,or in extreme cases death may result.The physical signs of acute dilatation of the heart rarely present much difficulty in their recognition.They are a feeble,fluttering diffused impulse;an increase of cardiac dulness to the left if the left ventricle is dilated,to the right of the sternum if the right ventricle is affected;and weak, rapid,indistinct heart sounds.The diagnosis is generally easy.The most prominent symptoms are extreme dyspnoea; weak,irregular,intermittent cardiac cation;and cerebral disturbances,with loss of consciousness in certain cases.Examination of the heart reveals a feeble and diffused impulse,an area of dulness to the right of the sternum,-for,except in cases of previous cardiac disease,the right ventricle isusually affected,-and feeble, indistinct heart sounds.It is sometimes impossible to learn at first whether serious dilatation has occurred or not,and we must wait until the effects of perfect rest can be observed before the diagnosis can be made. If actual dilatation has not occurred,the symptoms will pass away entirely,or will return in a milder degree upon exertion for a limited time afterwards.If actual dilatation has occurred,the symptoms will continue until death ensues,or until such time as may be

necessary for an hypertrophy sufficient to overcome the dilatation to develop.When acute dilatation is due to rupture of an aortic cusp,a diastolic basic murmur will be heard in addition to the signs already enumerated.

The symptoms of acute myocarditis are indefinite, and are usually obscured by the primary disease.There may be vague sensations,such as constriction or pressure,referred to the cardiac region,and occasionally even decided pain.The most important symptoms,however,are those indicative of cardiac weakness.Arrhythmia is a frequent and significant symptom when occurring in the course of rheumatic fever.The pulse is generally accelerated,and there may be paroxysms of great rapidity,the pulse-rate cometimes reaching 200 per minute.Dyspnoea and cough,with anxiety and general depression,may occasionally be prominent symptoms.Sudden dilatation of the cavities may supervene,and occasion extreme disturbance of the circulation.Endocarditis and pericarditis, whether preceding or following myocarditis,mask its symptoms,but in such cases the disturbance of the cardiac power is greater than would occur in uncomplicated endo- or pericarditis.Suppurative myocarditis may give rise to various embolic manifestations when the abscess ruptures into the heart.In such cases purpuric and pustular skin eruptions and splenic enlargement have occasionally been noted.The fever present in the primary disease usually rises somewhat with the development of myocarditis,and may be septic in type in the case of abscess.On auscultation the rapidity and irregularity of the action of the heart will be noted,and the first sound is remarkably weak.Murmurs are frequently developed as a result of dilatation and relative insufficiency of the valvular segments or of relaxation of the muscle at the orifice.The most frequent is a systolic bruit indicative of mitral regurgitation.Dilatation of the heart may occasion increase of the cardiac dulness.The diagnosis can never be made with certainty,but signs of great weakness of the heart,with a weak first sound or a systolic murmur,would point to the existence of the cardiac complication.The suppurative form may be suspected when the heart grows suddenly weak in the course of the parent disease,and if embolic manifestations supervene the diagnosis would be more certain.It is quite common to diagnose the condition during life and at autopsy find the myocardium unaffected. Should the myocardial inflammation become chronic the enlargement of the left ventricle is sometimes detected by the outward and downward displacement of the apex beat and by increase of the cardiac dulness in the same directions. The heart sounds at first may be clear and loud,but soon become decidedly weak and muffled.The tension in the lesser circulation is often elevated,and in these cases the second sound is accentuated,or even reduplicated,in the pulmonary region.A systolic murmur of mitral insufficiency may result from contraction of the papillary muscles and chordae tendineae or from dilatation of the ventricle and the valvular orifice.It is heard at the apex and towards the left axilla,but rarely has the wide transmission of the murmurs of organic mitral regurgitation.The action of the heart may be extremely irregular,and the sounds of varying loudness.The irregular rhythm called bruit de galop is occasionally heard,but is more properly attributable to the secondary

dilatation than to the indurative myocarditis.In many cases no symptoms are observed,and the fibroid disease of the heart is discovered at autopsy.The latency of these cases is due to the compensatroy hypertrophy of the heart and to the absence of disturbance of the innervation of the organ.Usually,however,failure of cardiac power to maintain the circulation is sooner or later manifested,though the symptoms vary so widely in different cases as to deprive the disease of any definite characters.Dyspnoea on the slightest exertion is frequently an early symptom,and may be accompanied by palpitation and other subjective indications.Constriction or pressure at the heart is very commonly experienced, and nor rarely there is decided pain of anginal character.Throughout the entire disease paroxysms of angina pectoris may be the only symptoms calling attention to the heart.In the intervals the patient may seem completely well,but the disturbances of the action of the heart generally persist after the anginal paroxysm has subsided.Cardiac arrhythmia is very common,and may assume any of its various forms.Perhaps the most common is slight intermission with inequality of the successive beats of the pulse.Cases are,however,at times observed in which the rhythm is constantly regular.The pulse-rate is generally decreased,and is frequently reduced to forty or fifty beats per minute.An extreme case is recorded in which the rate was said to have been but eight in the minute.In some instances,in which slowing of the pulse is constant and marked,patients complain of great palpitation during anginal attacks, even when the heart beats are far below the normal frequency.Sudden failure of the cerebral circulation is denoted by attacks of syncope,which come on without warning or more frequently when the patient has been exhausted by undue exertion.More rarely persons suffering from fibroid heart are prostrated by pseudo-apoplectic attacks,coming on after some mental or physical excitement,and sometimes leading to sudden death.Such attacks may occur in persons who have experienced no previous symptom of cardiac disease,and are easily mistaken for manifestations of cerebral haemorrhage.When the cavities of the heart begin to dilate,the evidences of failing circulation become more pronounced.Very often paroxysmal dyspnoea or cardiac asthma is observed.Later congestive enlargement of the liver,decreased excretion of urine,and derangements of the gastro-intestinal tract denote the progressive failure of the cardiac power. Throughout the entire course of the disease the patient may present great weakness and cloudiness of the mind, and in the latter periods may become either extremely somnolent or wakeful.Some of the cases are characterised by delirium and chronic mania.

BLOOD-VESSELS.

Acute arteritis has been described by Legroux,the intima immediately underneath the endothelium being the site of the inflammatory process.One of Barre's rheumatis fever patient s developed acute arteritis of the tracheal artery,and the literature contains other instances.Perivascular fibrosis is sometimes seen in subacute lesions which last a long time;and this fibrosis by constricting the lumen of the tiny arterioles and capillaries may interfere with the circulation in the part,-synovial membrane,pericardium,endocardium,etc.,-

and so give rise to a chronic process, which may or may not result in a fibrosis of the arterio-capillary kind. Rheumatism sometimes produces phlebitis, which is manifested by the usual symptoms of the condition, and is most frequent in the case of the lower extremities.

NERVOUS SYSTEM.

The nervous manifestations of rheumatic fever are of great importance and no little frequency; they are due either to functionaldisorder or very rarely to obvious organic lesions of the nerve centres or their menbranes. The dominant functional disturbance may be delirium which is greatly the most frequent; or coma, which is fare; or chorea, very commonly observed in children; or tetaniform convulsions, which occur seldom per se. As a rule, two or more of these forms coexist or alternate with or success one another, and the grouping, as well as rhe variety, of the symptoms may be greatly diversified. Besnier (loc.cit.) reports that in one hundred and twenty-seven observations there were thirty-seven of delirium only, seven of convulsions, seventeen of coma and convulsions, fifty-four of delirium, convulsions and coma, and three of other varieties. Either with or without subsidence of the articular inflammation, about from the eighth to the fourteenth day of the illness, but occasionally at its beginning, or sometimes on the eve of apparent convalescence, in cases of rheumatic delirium the patient becomes restless, irritable, excited, and talkative; sleep is wanting or disturbed; some excessive discharge from the bowels or kidneys occasionally occurs; profuse perspiration is usually present, and may continue, but frequently lessens or altogether ceases; the skin becomes pungently hot, the temperature generally, but not always however, rising rapidly towards a hyperpyrexial point, and ranging from 104. to 111. F.; and transient severe headache and disturbance of the special sense sometimes obtain. This condition is called cerebral rheumatism, and later on, or fromthe outset in hyperacute cases, flightiness of manner or incoherence in ideas is quickly succeeded either by a low muttering delirium, twitchings of the muscles, violent tetaniform movements and general tremors, and a condition perhaps of coma-vigil, or by an active, noisy, even furious, delirium. The articular pains are no longer complained of, and sometimes the local signs of arthritis also quickly disappear; but neither statement is uniformly true. The pulse becomes rapid, prostration extreme, semiconsciousness or marked stupour gradually or rapidly supervenes, the temperature continues to rise, the face, previously pale and fulshed, becomes cyanotic, and very frequently death ensues, either by gradual asthenia or rapid collapse, often preceded by profound coma or rarely by convulsions. Deep sleep often precedes prompt recovery. The duration of the nervous symptoms varies from one or two, or more usually six or seven, hours in very severe cases, to three or four days in moderate ones, or occasionally seven, eight, or sixteen or twenty-nine days in unusually protracted cases. In the last-mentioned, however, the delirium is not usually constant, and frequently disappears as the temperature falls, and recurs when it rises. Moreover, a rapid and extreme elevation of temperature is frequently wanting altogether. No real distinction can be established between these protracted cases of rheumatic delirium and so-called rheumatic insanity, in which occur prolonged

melancholia,with stupour,mania,hallucihations,illusions, etc.,often associated with choreiform attacks.This variety may be of short duration or continue until convalescence is established,or mayrarely persist after complete recovery from the articular disease.Coma may occur in rheumatic fever without having been preceded or followed by delirium or convulsions,although it is very rare;and,like delirium,it may obtain without as well as with peri- or endocarditis,or hyperpyrexia.It usually proves very rapidly fatal.In Priestley's case(Lancet, ii,1870,467),an anaemic woman of twenty-seven,during a mild attack of acute rheumatism,one night became restless;at 5 a.m.the pain suddenly left the joints;apparent sleep proved to be profound coma,and at 6 a .m. she was in articulo mortis.Southey(Trans.Clin.Soc.,xiii, p.29)relates the history of a girl of twenty who,without previous delirium or high temperature,suddenly became completely unconscious,and died in half an hour.One of Wilson Fox's(The Treatment of Hyperpyrexia,1871,4) cases had been completely comatose,and was apparently dying nine hours after the temperature had rapidly risen to 109.1.F.,when she was restored to consciousness by a cold bath and ice to her chest and spine.Convulsions of epileptiform,choreiform,or tetaniform character frequently succeed the delirium,but in exceptional cases they occur independently of it,and may even prove fatal. In addition to the choreiform disturbances which occur in connection with delirium,stupour,tremor,etc.,in cerebral rheumatism,simple chorea is frequently observed as a complication or as a sequela,or even,as an antecedent,of acute articular rheumatism,and they occasionally alternate in the same patient and in the same family.Chorea is perhaps most frequently seen in mild cases and in the declining and convalescent stages of rheumatic fever,and,while very common in childhood and adolescence,from five to twenty,it is very rare in adult life.The post-mortem examination in cases of these functional disturbances of the brain in rheumatic fever reveals either quite normal macroscopic appearances,or more frequently,especially in rapidly fatal cases, general congestion of the pia mater,and to a less degree of the cerebral substance,or in more protracted cases a greater or less increase of transparent or opalescent serum in the subarachnoid space and ventricles. The serum may be slightly or deeply tinged with blood. If the serous or sero-sanguinolent effusion be considerable,the encephalic mass or portions of it may be anaemic. But besides these conditions,which are commonly observed in many other febrile affections,and which are probably only concomitants of the functional disturbance arising in the advanced stage of acute articular rheumatism,certain organic affections of the nerve centres or their membranes occasionally occur in this disease, and are plainly the cause of the cerebral disturbance observed during life.Cerebral meningitis,although very rare as a complication of acute articular rheumatism, except in certain hot climates,like that of Turkey, does occur,and lymph or pus is found,usually over the convexity of the brain,but sometimes at the base and down the cord.The symptoms of rheumatic cerebral meningitis are very like those of rheumatic delirium;vomiting,and even,but less frequently,pain in the head,may be absent,while hyperpyrexia may coexist,although not

necessarily present.Should the pulse from being frequent become slow and irregular,and any paralytic symptoms ensue,meningitis may be suspected.In some of the cases the meningitis is the consequence of ulcerative endocarditis and embolism of the cerebral vessels,-that ulcerative endocarditis frequently produces meningitis is illustrated by Osler's(Trans.Internat.Med.Congr., 1881,i,344)cases,four of seven of which were complicated with purulent meningitis,- but with others it obtains without endocarditis or any purulent formation elsewhere than in the meninges,or the latter may promptly follow the disappearance of the former,as though a metastasis of morbid action had taken place;instances have been reported by Ramsey(Med.Rec.,1881,i,p.9)and others. In not a few cases,according to Ollivier,Ranvier,Behier, and others,although the macroscopic signs of meningitis are absent,the microscope detects proof of its presence in the existence of an increased number of vessels,fatty granulations on their walls,proliferation of nuclei and capillary extravasations - histological conditions identical with those found in the minor degrees of rheumatic inflammation of the joints.Embolism of the cerebral arteries,producing meningitis,or more frequently softening of the cerebral substance or haemorrhage,or proving fatal before necrobiosis has time to set in,is an occasional complication of acute articular rheumatism.There are cases on record in which,during a first attack of rheumatic fever complicated with endocarditis,the patient became suddenly hemiplegic and aphasic,and died twelve hours or so later.In a girl of thirteen,the subject of acute articular rheumatism complicated with ulcerative endocarditis,right hemiplegia suddenly occurred,and at the autopsy Bristowe found an embolism in the left middle cerebral artery and a softened area in the left corpus striatum.Bradbury(Lancet,1870,ii,148)reports a primary acute rheumatism with endocarditis,delirium, and coma,but without paralysis,in which a plug was found in the right middle cerebral artery,but the brain was in a normal condition.Another case(ibid.,1882,i,p.605), in the eighth week of rheumatic fever,there occurred embolism and right hemiplegia;the patient died,and at the autopsy there were found large vegetations on the cardiac valves and obstruction in the middle cerebral artery.Very much the same observations are applicable to the disturbances of the spinal cord and its envelopes in acute articular rheumatism as have been made with reference to those of the brain and its coverings.They may exist with or without any alteration of the cord or membranes to which they can be reliably referred;that is to say,they may be simply functional in the peculiar sense in which that word is now understood,or they may be connected with obvious structural changes,and chiefly with those indicating inflammation of the membranes or substance of the cord.The spinal symptoms may precede the articular affection,but generally appear after it. They closely resemble sometimes those of idiopathic tetanus,as shown in the cases reported by Bright(Trans. Med.-Chir.Soc.,xxii,4),Mann(Med.Rec.,1875,38),and Bouilland(Treatise on Diseases of the Heart,1872,i,9), or of spinal meningitis,or of myelitis,or of meningomyelitis;and in the last case,along with severe rachialgia,muscular rigidity,cutaneous and muscular hyperaesthesia,and neuralgic pains,there will occur numbness and more or less paralysis of the lower extremities,bladder,

and rectum - paraplegia.These spinal disturbances may or may not be accompanied by hyperpyrexia,and when simply functional they are usually less severe and persistent,have a greater tendency to alternate one with another and with the articular affection,and are more amenable to treatment,than when due to those very rare complications of rheumatic fever,spinal meningitis or meningo-myelitis.The inflammation may involve both the cerebral and spinal membranes at the same time.The causes of these disturbances of the nervous system,when not attributable to appreciable lesions,such as congestion,inflammation,haemorrhage,embolism,thrombosis,and softening,are not established.The following appear to be reasonable conclusions in the light of present-day knowledge.The most constant condition,and without which these cerebral symptoms very seldom arise,appears to be some susceptibility or vulnerability of the nervous system,inherited or acquired,rendering it apt to be disturbed by influences which less susceptible centres would successfully resist.Trousseau(Clin.Med.,Syd.Soc., 513),who has especially advocated this view,considered intemperance in the use of spirits to be a frequent cause of this nervous predisposition.Accepting this neurotic predisposition as the factor generally present when acute articular rheumatism is complicated by disturbances of the nerve centres,we may inquire as to the nature of the circumstances in the disease capable of developing into activity the predisposition in question. One of these conditions if the existence of acute pericarditis,or of endocarditis,or of inflammation of the lungs or pleura.Probably hyperpyrexia acts in some cases as an exciting cause of the nervous phenomena;for while the delirium preceded the hyperpyrexia in six cases(Abstract Report upon Hyperpyrexia in Acute Rheumatism,- Brit.Med.Jour.,1882,p.807),it accompanied it in nineteen and followed it in ten;and the nervous symptoms disappear when the hyperpyrexia is removed by the employment of cold,and recur with the return of high temperature.The phenomena of sunstroke and heat-apoplexy prove that a high temperature is capable of producing convulsions and coma.That these grave cerebral disturbances are so infrequent in rheumatic fever,- obtaining in about three or four per cent.only,- is probably owing to the usual moderate range of temperature and the rarity of hyperpyrexia in the disease.Still,when hyperpyrexia is a disturber of the cerebrospinal function,too much importance must not be attached to it;for not only does such disturbance very frequently precede the hyperpyrexia,but there are many facts indicating that the hyperpyrexia is itself very frequently,like the delirium, tremor,and coma which precede or accompany it,but a consequence of ~~xxx~~ disorder,usually of a paralysing kind,of the nerve centres.It has been met with in lesions of the pons,in tetanus,in injuries of the cord, in some cases of non-inflammatory softening of the brain and of cerebral haemorrhage;that is,in a class of affections not belonging to the specific fevers,but to those directly disturbing or destroying the functions of the nerve centres.And cases of acute rheumatism do rarely occur in which a very high temperature is not accompanied by cerebral disturbances.Sibson(loc.cit.) quotes two such,one of which with a temperature of 110.8.,was only restless and talked when asleep,and the

other,with a temperature of 106.3.F.,presented only vomiting and dyspnoea.Cardiac inflammation was absent in both.Da Costa(Amer.Jour.Med.Sci.,1845,p.36,Case xi) relates one in his interesting paper upon cerebral rheumatism in which neither cerebral nor cardiac symptoms were observed,though the temperature was 110.F. The large number of cases which have from time to time appeared in the literature in which grave cerebral symptoms have obtained in rheumatic fever at ordinary febrile temperatures,while they prove that hyperthermia is not an essential condition productive of such symptoms,require to be explained.Some such,no doubt,have been instances of marked predisposition,so that a moderately febrile temperature or some complication sufficed to disturb the brain,as we see in typhoid and other fevers,in pneumonia,etc.If there be a rheumatic poison, which has not yet been absolutely proved though rendered highly probable,it may,in predisposed persons,produce the cerebral symptoms.Maclagan's(Rheumatism:Its Nature, Pathology,etc.,1881,287)argument,that such poison should produce inflammation of the nerve centres if it acted directly on them,is not convincing.It need not necessarily produce similar alterations in serous or synovial membranes and in nervous tissues.Many toxic agents disturb,and even suspend,the cerebrospinal functions,and leave no appreciable changes in them.These cases scarcely prove that there is something peculiar to rheumatic fever which tends to disturb the nervous centres;for while such disturbance is comparatively rare in that disease,it is observed frequently in many other febrile affections,notably in typhus,scarlatina, and smallpox.And as in these,so in acute articular rheumatism,it is more often observed in the severe than in the mild cases,as though it were a part of the systemic disturbance incident to the febrile affection and largely proportionate to the intensity thereof.Nevertheless,there is something special in acute rheumatism which perhaps has to do with the occurrence as well as the severity of the cerebrospinal symptoms and of the hyperpyrexia,namely,the long duration and severity of the pain,and the number and importance of the parts,in addition to the articulations,which are one after the other or simultaneously involved in severe inflammation - pericardium,endocardium,myocardium,lungs,pleura,etc. Perhaps in no other acute febrile disease are so many distinct and important organs involved in inflammation at the same time or in rapid succession;and it is not strange to observe such great depression,exhaustion,or disturbance of the nervous functions therein.

RESPIRATORY SYSTEM.

TONSILS.

The occurrence of tonsillitis is one of the features of innumerable cases of acute articular rheumatism and known to even the writers of antiquity.There is evidence in favour of the theory that the throat is one of the channels by which the casual agent gains access to the system.In 1900 Paine and Poynton(loc.cit.) isolated from the tonsils a diplococcus very like the one they discovered in cases of endo- and pericarditis and arthritis,and which gave the like bacteriological reactions.Fritz-Meyer ,though he failed to find this microbe in rheumatic tonsillitis,is inclined to the opinion that the germ gains access in the way named.

His theory was later supported by such observers as Frisswell,who considers,however,that throat-cultures should not be accorded too much reliance.Though most of the arguments are in favour of a pathological relationship existing between rheumatism and tonsillitis,it must not be assumed that all cases of tonsillar inflammation are necessarily rheumatic.The theory in question has led to the excessive employment of the salicylates in tonsillitis,but the results obtained have often been far from satisfactory.Suppurative tonsillitis occurred in five of Mackenzie's forty cases of rheumatism.

LUNGS AND PLEURA.

Acute articular rheumatism is very commonly complicated with such respiratory affections as pleurisy, bronchitis or pneumonia.Adding Latham's(His Works,Syd. Soc.,i,98 et seq.),Fuller's(loc.cit.),Southey's(St. Barth. Hosp.Reps.,xv,14),Gull and Sutton's(Guy's Hosp.Reps., S.3,xi,434),Pye-Smith's(ibid.,xix,324),and Peacock's (St.Thomas' Hosp.Reps.,x,12-17)cases together,we have a total of nine hundred and twenty in which some one or more of the above pulmonary affections obtained in one hundred and nine instances,or nearly twelve per cent. A further analysis of Latham's and Fuller's cases shows that it is especially when rheumatic fever is complicated with heart disease that the lungs suffer;thus, pulmonary affections obtained in twenty-six per cent.of cases complicated with cardiac disease,and in only seven per cent.of cases free from that disease.It is more especially when pericarditis complicates acute articular rheumatism that pulmonary complications occur.Thus,these were found in only ten per cent.of cases of recent rheumatic endocarditis,in fifty-eight per cent.of cases of pericarditis,and in seventy-one per cent.of endo-pericarditis.The tendency which inflammation of the pericardium has to extend to the pleura probably partially accounts for the more frequent association of the pulmonary affections with rheumatic pericarditis than with rheumatic endocarditis;Sibson,it may be parenthetically noted found pleuritic pain in the side twice as frequent in pericarditis,usually accompanied with endocarditis(thirty-one in sixty-three),as in simple endocarditis(twenty-six in one hundred and eight). But the greater severity of those cases of rheumatic fever complicated with pericarditis or endo-pericarditis must also have a decided influence in developing the pulmonary affections.Pneumonia and pleurisy are very often double in acute articular rheumatism,and are often latent,requiring a careful physical examination for their detection.So suddenly does the exudation take place in some cases of rheumatic pneumonia that the first stage is not to be detected either by symptoms or signs.On the other hand,in some cases the absence of the typical signs of hepatisation,the want of persistence in the physical signs,and their rapid removal,and even in rare instances an onvious alteration between the pulmonary and the articular symptoms,suggest that the process often stops short of true hepatisation,and partakes rather of congestion and splenisation,with or without pulmonary apoplexy - a view which,from autopsical observations,has been occasionally confirmed by such observers as Sturges(Natural History and Relations of Pneumonia,1876,pp.70-78),Vasquez(Pleuro-Pneumonic Complications of Acute Articular Rheumatism,Thèse de

Paris,1878,pp.25-31),and Deveau(Dictionary of Medicine and Surgery,Vol.xxviii,p.443).Sometimes active general congestion of the lungs is observed in rheumatic fever, and Ball(Acute Pleurisy,1866)says that he has seen it prove fatal in five minutes,Aran(Quoted by Vasquez,-loc.cit.)in an hour and a half,from the invasion of the symptoms.The rheumatic poison frequently excites pleuritis,some of the characters of which - the suddenness with which free effusion occurs;the promptness with which it is removed,only perhaps to invade the other pleura,and then to reappear in the cavity first affected;the diffusion of the pain over the side and its persistence during the effusion;and its frequent occurrence with pericarditis,and in children with endocarditis;its little tendency to become chronic,and its marked proclivity to become double.It is often latent and unattended with pain.Sibson(loc.cit.)states that if in rheumatic pericarditis pain over the heart is increased or excited by pressure over the region of the organ,it may with an approach to certainty be attributed to inflammation of the pleura,The effusion is usually serous,but the formation of pus is sometimes observed.

ALIMENTARY SYSTEM.

Acute articular rheumatism sometimes,though comparatively rarely,finds clinical expression in affections of the alimentary canal.Such ailments are mainly observed in connection with the pharynx and intestine,some observers asserting that it is not uncommon for an attack of rheumatic fever to be introduced by fever, severe pain in the pharynx,and difficulty of deglutition. This is sometimes accompanied by painful swelling and difficulty of motion in the cervical muscles.A papular eruption is sometimes also visible for a short time upon the surface of the skin.These manifestations may be accompanied,or more generally followed,by articular pain and swelling.Sometimes inflammation of the tonsil proceeds to suppuration and the formation of abscess. The painful character of these pharyngeal inflammations is unusually severe,and is but slightly relieved by antirheumatic medication,and it may be that opiates alone will relieve the distressing symptoms.In rare instances rheumatic pain is experienced in the oesophagus during the course of acute rheumatism.Gastric pain and intestinal pain are commonly felt,but they are often overlooked in consequence of other neighbouring pains.Sometimes diarrhoea occurs,attended with pain of unusual severity. Ordinarily the liver remains inactive,and constipation is the rule during the evolution of the disease.Rheumatic peritonitis is a rare event,but is sometimes experienced in alternation with the articular manifestations.It is characterised by intense suffering,and is not infrequently fatal.In the opinion of some,rheumatic fever is a common cause of appendicitis,this old theory being revived by Yeo,Sutherland,Kelynack,and others.The first-mentioned of these observers has recorded a case which would seem to point very distinctly in that direction,although it was not entirely above criticism. The rheumatic theory of appendicitis is founded on the large amount of lymphoid tissue in the appendix wall making it the so-called"abdominal tonsil" of Ransohoff and Sutton,and also upon the good effect of salicylic acid and salts in catarrhal appendicitis;but it is questionable whether the connection between rheumatism

and appendicitis may not be equally explained by the frequency of intestinal disturbances in rheumatic or gouty individuals, and there can be no doubt that the use of salicylic acid and salol in cases of intestinal fermentation will prevent the latter by their action. Treves says that he has noticed, in a case of faecal fistula, that a foul discharge, owing to fermentation, was purified and rendered inoffensive in a few hours by the administration of salol. He has examined many cases of supposed rheumatic appendicitis, previously considered to be conclusive, and declares that the evidence is insufficient to warrant the acceptance of the hypothesis. Yeo's (Rheumatic Perityphlitis, Brit. Med. Jour., June 16, 1894) case is sufficiently interesting to merit reproduction in epitome: The patient, a young girl who had previously suffered from articular rheumatism, was suddenly taken ill with the symptoms of perityphlitis with high fever, and on the fourth day pains of the right knee, shoulder, wrist, and elbow made their appearance, while at the same time a systolic murmur was to be heard at the apex of the heart. These symptoms rapidly passed away on the administration of salicylic acid, but a few days later pain and tension reappeared in the right iliac fossa, and on palpating here a gurgling could be felt. This attack was also promptly controlled by salicylic acid. Yeo named this affection, after the analogy of those rare cases of rheumatic peritonitis, a rheumatic perityphlitis. It should, however, be noted that no tumour could be demonstrated in this case, and therefore the diagnosis of perityphilitis does not seem at all clear. In two other cases, reported from Brazil, in which an appendicitis was complicated with polyarthritis, we miss any direct casual relation, and there seems to be no reason why both affections might not have accidentally existed at the same time. The same applies to similar cases which Sutherland (Lancet, Aug. 24, 1895) gathered from the literature, and on the strength of which he comes to the conclusion that under certain circumstances appendicitis and rheumatism may be dependent on a third unknown poison, which may produce inflammation. Spillmann and Ganzinotti (Encyclopaedia Dictionary of Medical Sciences, T. 23, p. 310) collected fifteen cases of this kind, all of which, however, as Treves justly remarks, will not bear strict criticism. It cannot, however, be denied that appendicitis occasionally follows a cold. If every cold therefore be referred to some rheumatic or infectious cause, we may correctly speak of an infectious cause of appendicitis, one due to the sudden action of an external agent, and not to the bacteria. If any relation exists really between appendicitis and rheumatism, it would be in the highest degree surprising that this has not been discovered before, in view of the extraordinary frequency of the latter. The same is true of the view expressed by Goluboff (Appendicitis as an Epidemic Affection, Berl. klin. Woch., 1897, No. 1), who, because of the more frequent occurrence of appendicitis at certain seasons of the year, looks upon it as an epidemic affection like rheumatism sometimes is. Some of the eighteenth-century observers associated rheumatism with dysentery, this traditional idea finding support in the authority of Trousseau, who described a rheumatic form of that disease. Cambay, Quinquand, Dewevre, Dutruleau, Delioix de Savignac, Bérenger-Féreaud, and other formally admit a relation of cause and effect

between dysentery and the arthropathy which sometimes supervenes in its course.The articular symptoms may be observed during the evolution of acute dysentery during the first or second week;ordinarily,however,they appear during convalescence.In some cases it is polyarticular rheumatism,accompanied by or preceded by vague pains in the limbs or muscular pains,either fixed or generalised; this rheumatism runs its course concomitantly with the dysentery,disappearing with it or before it.In other cases the rheumatic affection is stubborn,being fixed from the start in one single articulation;or,as happens more frequently,it is localised in one or two,after having affected successively the others.This arthropathy develops as a rule during convalescence;it may also be observed during the evolution of the disease,coinciding with a suppression of the dysenteric flux.The pain is dull,without exacerbations;the local temperature is not modified,the teguments retain their normal colour;the periarticular tissues become tumid.The presence of liquid has been rarely observed in the synovial sacs.Despite its slow and prolonged evolution,the prognosis of the articular affection is favourable,as it always terminates without ankylosis,and without suppuration. These articular symptoms of dysentery would seem to be more frequent in temperate than in hot countries.

EYE.

Rheumatism of the eye seldom occurs in connection with acute articular rheumatism,but is sometimes seen in chronic rheumatism and pseudo-rheumatism.There is, however,nothing in the characters of the inflammatory lesions by which they can be distinguished from simple inflammations unconnected with a rheumatic cause.Rheumatis fever usually spares both the eyeball and its appendages.Iritis rarely occurs with it,but cases oc conjunctival congestion without muco-purulent discharge have been reported.One is compelled,however,to recognise the use of the term rheumatism as applied to an extensive group of symptoms which are probably dependent on the same causes with articular rheumatism.Sudden exposure to cold and overmuscular exertion are the chief exciting causes.Some of the diseases ascribed to the chronic type of the disease are iritis,episcleritis, scleritis,keratitis,orbital cellulitis,optic neuritis, choroiditis,ocular paralysis,glaucoma,and opacity of the vitreous.Iritis is the most important of them all as regards frequency.Some observers say that iritis furnishes from two to four per cent.of all ophthalmic cases,and that syphilis and rheumatism are causative factors of this disease in ninety per cent.in the proportion of rheumatism thirty,and syphilis sixty.There may,in the absence of syphilis,be a history of acute or chronic articular rheumatism,symptoms of lumbago, neuralgia,sciatica,torticollis,pains in the articulations or fasciae,or sensitiveness to changes in the weather.Many cases are subject to relapses of iritis in spring and winter;seasons of the year when rheumatic affections are most prevalent.Some cases of iritis alternate with rheumatism in other parts,and some recur with the swelling of the joints.Next in point of frequency comes a superficial form of scleritis,episcleritis,which consists of a circumscribed inflammatory nodule generally on the temporal side.It is not movable over the sclera,but firmly attached and of a reddish-

violet colour.This condition can never be looked upon merely as a local disease,but must be regarded as the manifestation in the eye of some systemic derangement. The large percentage,if not all of them,are associated with the rheumatic diathesis.One of the rarest,but nevertheless the most serious,rheumatic affection of the eye is deep scleritis.Here the circumcorneal congestion is more general,showing extensive bluid-red discoloration.It is a chronic disease and destructive to sight by the formation of deposits in the cornea and staphyloma of the sclerotic.

MAMMARY GLAND.

Inflammation of the mammary gland is said by Still to occur in children,and he reports two cases of rheumatic fever complicated with mastitis.

LYMPHADENITIS.

Rheumatic fever is accompanied,in severe cases by great enlargement of the mediastinal glands;and the cervical glands are now and then greatly enlarged in tonsillar cases.A general enlargement of the lymphatic glands is,however,of exceptional occurrence.

EAR.

While there are no satisfactory reasons for doubting that rheumatism sometimes causes middle-ear disease, as a matter of fact we do not possess as yet any data which satisfactorily establish the fact.The footplate of the stirrup becomes immovably fixed in the oval window,and as the result of this the hearing power becomes reduced to almost nothing;and what is more likely than rheumatism(and also gout)to have caused such a rigidity? If a similar immobility occurs in any other joint in the body we at once suspect such an affection as rheumatism as having been the cause of the lesion,and it seems that we may with equal propriety suspect this disease of having been the cause of a similar immobility in the stapedio-vestibular joint.Another way in which this disease may at least aggravate a catarrhal inflammation of the middle ear is when the affection gives rise,as it sometimes does,to an active and generally very stubborn naso-pharyngeal catarrh,or at least intensifies an already existing catarrhal affection of this region, and so eventually involves the middle ear.

PAROTID GLAND.

Trousseau mentions the occurrence of articular pains in mumps which were similar to those observed in scarlatinal rheumatism.Jourdan,in a series of sixty-one cases,observed four of articular pains in the shoulders, elbows,and wrists.These articular affections,which are unmarked by swelling or effusion into the joints,usually disappear in the course of a few days.In some of the reported cases there is reason to suspect that true rheumatism was the underlying condition.Thus,Rilliet has seen rheumatic fever follow an attack of parotitis in two brothers,but one of these had suffered from true rheumatism for some years previously,and the mumps served only as an exciting cause to awaken into activity the old disease.In other cases,however,it seems as though the attack of mumps really determined a sort of infectious pseudo-rheumatism.A few joints only are involved at a time,fixation in consequence of the arthralgia occurs,the process passes along the synovial sheaths,and invades the bursae,- the prepatellar bursa in Bergeron's case,- there is moderate fever,

hydrarthrosis occurs, etc. This form of rheumatism usually ends in resolution, but suppuration may occur in some cases. Sometimes the rheumatic symptoms precede the occurrence of the parotitis; Catrin in one case sound the exudate to contain a diplococcus.

RHEUMATISM AND SCARLET FEVER.

Articular affections are not very common in scarlatina, but there sometimes occurs in certain cases or certain epidemics what has been termed scarlatinal rheumatism, which is a synovitis developing usually during the latter part of the period of efflorescence or at the commencement of dequamation. It attacks most often the larger joints, such as the ankle and wrist, but may be seen in the fingers or any other articulation. As a rule, there is little swelling, but the joint is painful and tender. Some believe that this affection is really rheumatism complicating scarlet fever, but I do not believe that this assumption is necessary to explain the phenomena presented, and prefer to regard scarlatinal rheumatism as due to the action of the poison of the disease after which it is called. It is maintained by some authorities that the presence of scarlatinal rheumatism increases the liability to valvular disease of the heart. Generally the inflammation of the joints subsides in a few days, but in rare instances a suppurative synovitis develops, with destruction of the joint. A fatal pyaemia sometimes terminates the life of the patient when the large articulations are involved.

RHEUMATISM AND RELAPSING FEVER.

Patients suffering from relapsing fever sometimes complains of pains in the joints, and occasionally we find on examination redness and swelling of the affected articulations. The most frequent seat of these pseudo-rheumatic affections will be, in order of frequency, the upper extremities (fingers, wrists, elbows, shoulders), and then the knees; but this order may be reversed. The character and course of relapsing fever in which this complication is encountered do not differ in any respect from those of an uncomplicated attack. The articular lesions generally terminate in recovery, and they respond quickly to the exhibition of the salicylates.

RHEUMATISM AND CHOREA.

Chorea may complicate rheumatism, but it is more often observed as a sequel, occurring in a subject of rheumatism between the rheumatic attacks. There has been much discussion as to the real relation existing between the two diseases, some holding that all chorea is evolved from a rheumatic diathesis, and that endocarditis occurring in a case of chorea is a manifestation of rheumatism, although no arthritis develops at any time. Duckworth has estimated that fully seventy-eight per cent. of cases of chorea are of true rheumatic origin. German writers, however, attach much less importance to this association, and Steiner found but four cases of rheumatism among two hundred and fifty-two of chorea. Osler (Practice of Med.) found the percentage of association not above twenty-one. The two affections are apt to occur at about the same season of the year, and chorea often follows within a month after a rheumatic attack. The subcutaneous fibrous nodules already described may be present in chorea, but pericarditis belongs especially to rheumatism, while endocarditis, leaving permanent valvular lesions, is quite common in both diseases.

SECONDARY RHEUMATISM.

The use of this term calls for special explanation. It has been applied to certain forms of articular disease which occur as a consequence of particular infective maladies. Of these the most common varities are the arthropathies that sometimes follow mumps, scarlet fever, typhoid fever, relapsing fever, puerperal fever, pyaemic, dysentery, syphilis, and gonorrhoea - some of which have receeved short notice above. Until the discovery of actual cause of rheumatism it will be impossible to speak positively regarding the exact relation that exists between such articular affections and genuine rheumatism. It is probable, however, that their ultimate causes are different, but that the local manifestations of the action of such various causes possess a degree of resemblance that is based upon the common identity of the tissues in which the local changes are exhibited. It is maintained strenuously by innumerable observers that there cannot be any other point of union between scarlatinal or gonorrhoea rheumatism and genuine articular rheumatism. This view is rendered still more probable by the failure of the antirheumatic remedies, like the salicylates, when administered in cases of gornorrhoeal or sclarlatinal rheumatism; the characteristics of these pseudo-rheumatic affections can be therefore most appropriately considered in connection with the diseases from which they respectively originate, though gonorrhoeal arthritis will later on be specially described in observance of an old-time custom when dealing with the rheumatic taint.

PREGNANCY AND THE PUERPERIUM.

It is necessary to consider briefly the influence of pregnancy and the puerperium upon rheumatic fever, owing to the affirmation of some that both tend to give rise to a malignant form of the disease. It is well-known that pregnancy aggravates chorea, and in not a few cases (French and Hicks,- Practitioner, Aug., 1906) it would seem that the patients have presented a history of rheumatism or chorea in former years. Rheumatic inflammation of the heart has also been known to rejuvenate and terminate the life of the patients by malignant expression; and Payne and Poynton, and Shaw tell us that some of the lower animals, after recovering from acute rheumatism complicated with endocarditis, quickly died from malignant and aortic endocardial inflammation when they became pregnant and were infected with rheumatism again.

COURSE AND DURATION.

Great variation may be exhibited in the course and duration of rheumatic fever. This may be due to several circumstances, such as the severity or mildness of the joint affection, as well as of the constitutional disturbance; the presence of not of complications; the state of the health of the patient about the time of the attack, and probably the existence or not of a proclivity to the disease; and whether the disease present the continued or the relapsing type. Generally speaking, when the constitutional symptoms are acute, the skin hot, the perspiration free and very acid, the urine of high density and colour and acidity, and several of the articulations are swollen and very painful: when no

thirteen new cases and twelve of those published in their two previous communications, they conclude that rheumatic fever uncomplicated by any very severe cardiac affection tends to run its course in nineteen days, calculating from the time the rheumatic symptoms first set in to their termination (Trans.Med.-Chir.Soc., lii, 82). Yet an analysis of the twenty-three of the forty-one cases contained in their first series (Guy's Hosp. Reps., xi, 435) respecting which the duration of the rheumatic symptoms before admission and from admission to complete convalescence is given, shows that the period occupied from the setting in of the rheumatic symptoms to convalescence was in the thirteen male subjects twenty-five days, and in the ten female forty-two days, or, including both sexes, the average duration was about thirty-two days, that is, some six days longer than Flint observed. As Gull and Sutton had especially pointed out the class that tends to assume acute characters and recover more quickly than any other, and the class that runs a protracted course and tends to relapse, it is somewhat remarkable that they did not tabulate the cases belonging to those classes separately, and show distinctly their differences in duration and modes of convalescence. This has been attempted by Southey (St.Barthol.Hosp.Reps., xiv, and ibid., xv), but, unfortunately, his conclusions have not been confirmed by other observers. Russel (Essay on the Convalescence of Acute Articular Rheumatism, Paris, 1881, 66), in this connection, after studying ten rheumatic fever patients during their illness and until they had gained their usual weight, found that the time during convalescence occupied in regaining their weight previously lost was inversely proportional to the time that the illness lasted.

SUBACUTE ARTICULAR RHEUMATISM.

Charcot, Besnier, and Homolle were the first to describe under this heading an affection which closely corresponds with one variety of the disease called arthritis deformans; but here we may use the term to designate an affection characterised by articular lesions similar to those which are encountered in the acute form, their evolution is less speedy, cardiac and other visceral complications are less frequently observed, and convalescence is more protracted than in the acute disease. The local articular manifestations are less painful, the large joints are not invaded as often as the small joints of the extremities, but the affection is much more tedious and obstinate then when it assumes the acute form. The subsidence of local disorder proceeds very gradually, and is often imperfectly accomplished, so that stiffness of the joints and adhesion of the articular surfaces are more likely to persist as a permanent result of the disease. Visceral lesions are less frequently encountered than in acute articular rheumatism, but when they do occur they are quite as complete and as formidable as when they accompany the acute disease. There is no uniform relation between the intensity of the articular manifestations and the severity of the visceral lesions: a moderate degree of external disorder may be accompanied by the most dangerous alterations of structure in the heart and other internal organs. In like manner, though febrile symptoms are usually

insignificant,the blood becomes as completely impoverished as in acute articular rheumatism.The course of subacute rheumatism is quite continuous,though subject to partial remissions and moderate exacerbations:there are nothing like the rapid changes and migratory character of acute rheumatism.The duration of the affection is usually prolonged from six weeks to several months. The malady is not radically different from the acute form.The characteristic variations are founded upon individual peculiarities of constitution and temperament rather than upon differences pertaining to the cause of the disease.It must be admitted,however,that in many cases the symptoms and behaviour of the affection closely resemble those of the forms of rheumatism that are dependent upon a previous infection,such as gonorrhoeal rheumatism.It is impossible to speak with decision regarding these matters in view of the present doubt as to the ultimate cause of rheumatism.It would appear that the process is an inflammation involving chiefly the synovial membrane,and to a less degree the cartilages,ligaments,tendinous sheaths,and in some cases even the bones and periarticular soft parts.The synovial membrane is more or less injected and reddened diffusely or in patches,especially when it forms fringe-like folds and at its line of union with the cartilage. It is somewhat thickened,opaque,and devoid of its satin-like lustre,and in somewhat protracted cases covered here and there with a thin,easily detached neo-membranous formation.Within the articulations will be found from a few drops to one or two ounces of a viscid,and generally turbid,pale,viscid,citron- or reddish-coloured fluid, like synovia,but more fluid,and containing transparent or semi-opaque gelatinous masses or albumino-fibrinous flocculi.The microscope reveals in the effusion large detached spherical epithelial cells in various stages of germination or of fatty degeneration,and a variable number of red blood-corpuscles and pus-cells.Very exceptionally the effusion is mixed with more or less true pus.In two out of the eight fatal cases reported by Fuller(loc.cit.),in which the joints were examined,pus in moderate quantity was found along with the other products in some,but not in all,of the inflamed articulations,and one of them was complicated with erysipelas,the other with sloughs over both trochanters.In very severe forms complicated with haemorrhagic tendencies the inflammatory products have contained a large proportion of blood.Cornil and Ranvier(Manual of Pathological Histology,Paris,1869,406)insist that even in slight cases of rheumatic arthritis the diarthrodial cartilage constantly suffers changes arising from nutritive irritation and proliferation of the cartilage cells. At first the cartilage loses here and there some of its polished hyaline appearance,and the microscope reveals a finely striated condition of its structure which gives it a velvety aspect.When the inflammation has been more severe and of longer duration,so that the deeper layers have been involved,the unaided eye will perceive local swellings in which the natural elasticity and resistance of the cartilage are impaired,and its surface is fissured or villous-like in appearance.True ulcerations of the cartilage are seen in certain rare cases of mono-articular rheumatism.The periarticular soft parts may be in some cases more or less congested

serious complication, and especially no severe cardiac affection, exists, and when the patient is endowed with a fair constitution and with organs not damaged by previous disease, the course of the fever is tolerably short and continuous, and the recovery more or less prompt. Amongst the most reliable evidences of approaching recovery in such cases if the tongue becoming clean and losing its red colour and the urine increasing in quantity, but containing a large proportion of solid matter, as indicated by a high specific gravity. On the other hand, a large proportion of cases run a more irregular and protracted course, and more or less marked relapses succeed real but temporary improvements, the local disturbance affecting fresh joints or reappearing in those previously attacked, and the general symptoms resuming renewed activity. The duration of the active symptoms in these cases is considerable, seldom under six weeks, and frequently occupying seven, eight, or more. In these protracted cases the symptoms, as a rule, are usually rather milder, the perspiration not as profuse or sour, the urine of less density and acidity, the articulation less hot and painful, than in the previously-described group. Sometimes, indeed, the perspiration and the urine are of neutral or even faintly alkaline reaction. It is not only the unexplained tendency to relapse which protracts these cases, but sometimes in addition an established proclivity to the disease (the rheumatic habit) or a condition of previous bad or generally defective health. Such cases occasionally pass into the subacute form, or the mild febrile symptoms gradually and finally decline, and the joints may either remain tender, swollen, and stiff some time longer, or these signs of recent inflammation may soon disappear and leave the articulations merely weak. Not a few cases of rheumatic fever embody several of the features of the two groups just described, and no definite course or duration of acute articular rheumatism can be stated on hard-and-fast lines. There has from time to time occurred considerable discussion regarding the course and duration of the disease. Flint (Amer. Jour. Med. Sci., July, 1863) was one of the first to study the natural history of the affection uninfluenced by active treatment, and he was followed, two, three, and six years respectively, later by Gull and Sutton (loc. cit.), who treated a series of cases without medicine, unless peppermint-water can be so regarded. The mean duration of Flint's thirteen cases from the date of attack to convalescence, excluding one in which pericarditis and pneumonia occurred, was a fraction under twenty-six days. It is unfortunate that the number of cases was so small, and that eleven of the patients were females, who appear to be especially subject to the milder and more protracted attacks of the disease. A larger number, with an equal proportion of the sexes, would probably have given a different result. Gull and Sutton narrate the natural histories of sixty-two cases, namely, of forty-one in their first series, of eight more in their second, and of thirteen more in their third. The average duration of the acute symptoms was, in the first series, eight days, in the second nine days, and in the third ten days, giving an average of about nine days for the duration, after admission to hospital, of the acute symptoms of acute articular rheumatism when there is no severe cardiac disease. In their third paper, based upon

and oedematous,and the tendinous sheaths,and even the bursae mucosae,inflamed and distended with inflammatory products like those in the articulations.Charcot(Clinical Lectures on Acute and Chronic Diseases,Syd.Soc.,1881,p. 148),holding the opinion that arthritis deformans is but a chronic variety of acute articular rheumatism, quotes Gurlt's statement that in acute articular rheumatism"the medullary tissue of the ends of the bones undergoes a great increase of vascularity,with proliferation of its corpuscles",and remarks that Hasse and Kussmaul have also referred to lesions of the bones and periosteum in that disease.But the condition of the osseous parts of the joints in acute articular rheumatism can hardly be said to be known,and it is unnecessary to speak with decision thereon.In subacute rheumatism the alterations in the synovial membrane,and especially in the cartilages just described,are likely to be more marked than in the acute form.It is not infrequently difficult to distinguish subacute rheumatism from other chronic articular diseases,from secondary rheumatism, from chronic rheumatism in its stage of exacerbation,and from subacute varieties of gout that involve the small joints.The effects of colchicum and the application of the uric acid test will often furnish the means for discrimination between rheumatic and gouty disorders,and the course and termination of the disease will distinguish it from genuine chronic rheumatism,while attentive consideration of the history will aid in the differential diagnosis between subacute articular rheumatism and other secondary articular affections.

MONO- OR UNI-ARTICULAR ACUTE AND SUBACUTE RHEUMATISM.

It is quite exceptional to find acute rheumatism invading a single joint to the exclusion of the rest;yet now and then a case occurs.It is perhaps impossible to be certain that such an arthritis is rheumatic unless some of the other symptoms or complications of articular rheumatism supervene,or unless it have succeeded a polyarticular rheumatism,which it very rarely does.Monoarticular rheumatism is very generally of the subacute type,and unattended with fever from the outset,or only a moderate pyrexia obtains for a few days;there is generally considerable effusion into the joint,with swelling,pain,and moderate local heat;visceral complications very seldom occur,but the local inflammation persists most obstinately for six or eight weeks or three or four months,and often leaves the joint tender,stiff, and weak for a long time or even permanently.In both the acute and subacute forms,before concluding that arthritis of a single joint is rheumatic,we must exclude the probability of its being traumatic,strumous, syphilitic,gonorrhoeal,neurotic,or,above all,of the nature of rheumatoid arthritis,which many such cases actually are.

ACUTE ARTICULAR RHEUMATISM IN CHILDREN.

Acute articular rheumatism,when it occurs in children,presents certain peculiarities,which may here be specially noted.The delicate tissues and organisation of a child show a more extensive area of disturbance under the influence of the disease than the stronger textures of the grown-up individual.Some phenomena,unquestionably associated with the rheumatic state in early life,such as chorea and the development of

tendinous nodules, for instance, are rare or altogether wanting in later years. Besides, in the rheumatism of childhood the chief and most characteristic phenomenon of the disease in adults, the articular affection, is not infrequently extremely slight, sometimes absent altogether, it being overshadowed or replaced by other phenomena occasioned by the same morbid principle which produced the arthritis. In children more than in adults it would seem that there are several manifestations connected wholly of partially with the rheumatic state which are not limited to the joints or to fibrous tissues or serous membranes, but involve occasionally also mucous membrane and skin, and even the central nervous system itself. Erythema, tonsillitis, chorea, pleurisy, tendinous nodules, may be the result of the rheumatic disturbance as certainly as arthritis or pericardial inflammation. They are found associated with articular rheumatism, and when they occur alone are met with especially in rheumatic subjects. These affections in certain instances must be regarded as manifestations of the rheumatic state, although they may be set up in other instances by other causes, just as articular inflammation or pericarditis, while usually rheumatic, may be due to scarlet fever, septicaemia, or pyaemia. They are not invariably rheumatic, but most commonly perhaps rheumatic. This series of rheumatic phenomena may occur in any order of sequence, in any combination. Any one of the phases may be absent, one only may be present, or two or three, or the whole series may be complete in the same patient. There may be articular affection only, for example, or there may be in addition pericarditis or endocarditis, or these may occur without any affection of the joints, or with chorea or tendinous nodules, or there may erythema or tonsillitis instead of any of these, or in addition to them. This is a common observation. The different manifestations, again, may occur not only in any order and combination, but separated by varying intervals of time, following one another in quick succession, or some appearing months or years after the rest. Thus, an endocarditis or a pericarditis or a chorea may occur first and alone, the articular affection long after. The various manifestations massed together in the case of adults tend to become isolated in the case of children, so that the whole phenomena are distributed over years instead of weeks or months, and the history of a rheumatism may be the history of the entire life of the child. The articular affection frequently not being a marked feature of the rheumatism of childhood, and it sometimes being altogether wanting, it cannot be looked upon as essential or typical as in adults. Endocarditis is perhaps the most constant rheumatic phenomenon observed in children, the articular affection being just as rare as it is frequent. In childhood the manifestations do not centre round the articular lesion, and when the latter is slight or wanting or has occurred apart, before or after, the endocarditis, or pericarditis, or pleurisy, cannot be well looked upon as secondary and dependent upon it. These inflammation, indeed, are just as much direct results of the rheumatic virus, whatever its nature, as the articular inflammation, so that any description of the disease must take them fully into consideration.

The etiology and pathology have already been

sufficiently considered;and in dealing with the symptomatology it is necessary to bear in mind that the articular inflammation,which is such a constant and striking feature of the adult disease,in childhood sinks into comparative insignificance,and is often entirely absent in an attack which is undoubtedly one of acute rheumatism.Moreover,many of the phases or manifestations of rheumatism which,considered from the adult standpoint, are generally regarded as complications or sequels of a central articular affection,appear in childhood as initial or chief phenomena.Endocarditis or pericarditis may appear first,or pleurisy,or chorea,or tonsillitis,or nodules,or an erythema,or an arthritis,and these may be grouped in any order,in any number,separated by varying intervals of time.In early childhood the tendency is to isolation and separation of the phenomena.These draw more closely together with the lapse of time;the disease tends to appear as a whole,instead of in disjointed parts;some features become accentuated,as the articular affection,others grow less constant and conspicuous with advancing age,as the tendinous nodules and chorea,and these finally disappear,except in rare instances,wwith the advent of adolescence.

The association of acute endocarditis with the rheumatic state in childhood is well known and plays a conspicuous part.It appears with the articular affection in the majority of instances,and a small proportion only of children escape it;if arthritis is present,we are pretty sure to have endocarditis;the Collective Investigation Statistics give in males seventy-two per cent of cardiac affection in children,as comparedwith forty-six per cent.in adults.But often the endocarditis appears alone,the sole expression at the moment of the rheumatic state,or is accompanied by the eruption of subcutaneous nodules so intimately associated with the evolution of valvulitis in early life,or by chorea or erythema.The affection is constantly overlooked,because the significant articular affection is slight or wanting;the child is a little wasred or feverish,but there is nothing to call attention to the heart,and thus an insidious inflammation of the valves progresses,and is probably not discovered until long after it existence is proclaimed by hypertrophy and dilatation of the organ.The endocardial inflammation is nearly always subacute,and is often protracted and relapsing;it subsides and then rejuvenates itself again.It attacks chiefly the mitral valve,but now and then the aortic valves suffer,and in exceptional instances they are alone affected.The first sign,and sometimes the only sign,of the valvular inflammation is a soft blowing murmur,usually systolic,at the apex.This may gradually disappear after a few weeks,or more often may increase rapidly in distinctness,so as to become loud and harsh in the course of a few days.Yet sometimes the murmur, even when mitral,may be functional,due to temporary relaxation of the papillary muscles and consequent imperfect closure and leakage;and this may disappear as strength and muscular tone return.Yet the fact that such murmurs appear,as a rule,in the earliest stage of the rheumatic attack,before serious debility of the cardiac muscle is likely to have occurred,points to its being due to valvular inflammation rather than to functional disturbance from paresis;and the disappearance of the

murmur should be referred to resolution of the inflammatory process and restoration of the valve to its normal state.A distinct mitral murmur is usually organic, indicative of endocarditis,and commonly persistent.An aortic obstructive murmur is in like manner an almost certain indication of endocarditis.An aortic regurgitant murmur is invariably organic.Another auscultatory phenomenon pointingto the development of endicarditis, and especially in childhood,is a reduplication of the second sound,audible not at the base,as in the doubling from increase in one arterial system as against the other met in the systemic obstruction of nephritis on the one hand and in pulmonary obstruction on the other, but at the apex only.This reduplication is sometimes accompanied by a diastolic murmur after the second of the two parts of the double sound.These signs may disappear;or more often they increase in intensity and gradually develop into the presystolic rumble.They are, indeed,the first signs of mitral stenosis,and certain indications of valvulitis going on,that is,the supervention of endocarditis.In spite of the development of these signs of endicardial valvular inflammation,there may be no rise of temperature,no quickening of the pulse,no distress,sometimes a pyrexia of one or two degrees,and some excitement and quickening of the heart's action. But the only certain sign of the commencement of endocarditis is that afforded by the alterations in the cardiac sounds.

The occurrence of pericarditis is very apt to be overlooked in the rheumatism of childhood,owing to the slightness of the associated articular symptoms and to its occasional occurrence entirely apart from them,and partly to the inflammation being generally subacute with slightly-marked symptoms which do not command attention.Pericarditis is met with in very young children even,although less often than in later childhood.West (Diseases of Infancy and Childhood,Ed.7,pp.556,557) describes a case in a child of seven months,with autopsical evidence of a previous attack at the age of four months.The phenomena of pericarditis have already been fully described,and need not be repeated;but there are special features connected with the affection as it is seen in childhood which claim our attention now. The disease is perhaps less likely to occur in a primary attack of rheumatic fever;like endocarditis,although it is at times very severe,it is comparatively rare;and it has a characteristic tendency to become subacute, chronic,and intermittent,to smoulder on and then become active again,with the advent perhaps of a fresh wave of articular inflammation ,or the supervention of chorea, or the appearance of a frsh crop of nodules.Although generally associated with arthritis,it may be the first and only sign of the rheumatic state and at the time of its occurrence,and be followed by articular inflammation at varying intervals;or it may be the last of the series of rheumatic events.Although not rare in the early period of the disease,it is most common,or at any rate most often observed,when the heart has already become greatly enlarged by hypertrophy and dilatation;and it is then most liable to set up fever and palpitation, with excited and turbulent and irregular action of the heart and quick pulse,sometimes excessively so,-from 120 to 160 even,- with cardiac pain,dyspnoea,restlessness,

and distress.Very possible,however,there may have been pericarditis before;it does not leave a record of its presence behind it,like endocarditis.This late pericarditis very often carries off the patient later on.In the early attacks,however,the general symptoms are usually limited,except in the rarer acute cases,to slight fever,with a moderately accelerated respiration and pulse.The physical signs of pericarditis in children are the same as in adults:friction audible over the preacordia,followed by dulness,increased in intensity and extending according to the amount of liquid or lymph effused,and subsequent distention and thickening of the pericardium.This is sometimes considerable when the inflammation has been repeated or persistent and extended to the connective tissue of the anterior mediastinum,causing the enormous thickening which we have elsewhere described.With this increase in dulness there is also some muffling of the heart's sounds over the central portion of the cardiac area,simulating that produced by effusion.From this it may be distinguished, however,partly by the less distinctly triangular shape of the area of dulness,but chiefly by the fact that the apex is displaced not upwards,as in serous effusion,and the heart's sounds at this point are comparatively sharp and clear.But as with general symptoms,however,so with physical signs;they are,as a rule,not pronounced in the primary attack.Instead of the marked changes described above,there is merely the double friction sound,lasting for alimited period,and disappearing as adhesion takes place,to be renewed perhaps and lead eventually to the more marked changes which later on occur.

According to Fisher(Brit.Med.Jour.,July 18,1896), in many cases of rheumatism the mitral murmur which so frequently arises is due to <u>simple dilatation</u> of the left ventricle causing mitral leakage,without any organic changes in the falps of the valves themselves,and analogous to the well-known pulmonary haemic murmur. There have been several cases recorded in which the only lesion found after death was dilatation of the cavities of the heart.But that this simple dilatation alone, without valvular inflammation is not a common source of mitral murmur is shown by the constancy with which persistent mitral bruit and evidence of organic valvular affection eventually ensue.Lees(Lancet,July 25,1896) has shown that dilatation does occur in the early stage of rheumatic fever,as evidenced by the increase in the area of cardiac dulness.He finds that this dulness may extend as much as two fingers'-breadths beyond the right edge of the sternum,the same distance beyond the left nipple-line,and upward to the level of the second left costal cartilage.This may be seen in cases free from pericarditis;and our author is inclined to regard the dilatation as a special result of the enfeebling influence of the rheumatic poison.He says that a similar condition may occur in chorea,and this has been confirmed by cardiac measurements taken by various observers.

<u>Articular inflammation</u> is a comparatively slight symptom in children;in some instances there may only a little tenderness and swelling of the knees or ankles or wrists,possibly limited to a single joint,or even less than this;there may be mere stiffness and tenderness on movement,recognised afterwards as rheumatic by

the occurrence of cardiac complications.In other cases the rheumatic inflammation is limited to tendons or their sheaths,as in stiff-neck,which is occasionally the only manifestation of genuine rheumatism.In some instances this gives rose to prolonged torticollis,where the rheumatic nature of the affection is shown by the previous articular trouble,as well as tonsillitis and cardiac symptoms.One of the most misleading manifestations of rheumatic affections of the joints or tendons is when it is limited to stiffness of the hamstring tendons at the back of the knee.Not infrequently,however,the articular symptoms are sufficiently marked to attract attention,and both wrists,knees,and ankles,and possibly fingers,present the typical appearance of acute articular rheumatism.The older the child,the more nearly does the affection conform to the adult type in this respect. One seldom sees a little child,unable to move,and bound hand and foot with rheumatic inflammation of the joints as in grown-up persons.They are stiff and tender,but the patient usually is able to move about.The articular affection migrates perhaps from joint to joint,and tends to change to the subacute form and to be characterised by relapsing tendencies.Further,the temperature differs in its range from that observed in the adult disease.It seldom is high,rarely above 102. to 103., except in the older children,more often 100. to 101.F.,and this febrile rise lasting only for a few days.This is the more striking because it is at variance with the general rise of temperatures in childhood,which tend to become more easily raised and to range higher than in later life.And, as anything like high temperature is rare,fatal hyperpyrexia is unknown,and one element of immediate danger is wanting.In accordance with what is noticed in connection with the temperature,the rate of the pulse is but slightly raised,unless there be accompanying cardiac or other inflammation.

Unless there are complication present and productive of hyperthermia and constitutional disturbance in severe cases,the tongue is seldom much coated or dry.The thickly-furred or dry brown tongue encountered in severe cases of adult rheumatic fever is never seen in children.

The urine is scarcely affected in the milder cases, but it becomes darker,acid,lithatic,and dense when the arthritis is severe and the temperature high.

The profuse acid perspiration ,which is so marked in the rheumatic fever of adults and constitutes one of its diagnostic signs,is wanting in the case of children. The sweating is very slight;the patient is never seen bathed in moisture soaking nightdress and pillow,and it is not possessed of a sour odour.Furthermore,children do not show the characteristic sudamina and miliary vesicles of adults.

We have seen that chorea is not without rheumatic association.Roger says that all chorea is of rheumatic origin,but this is obviously an exaggeration of facts, as other things may produce it.Nevertheless the arthritic diathesis is responsible for its occurrence in not a few instances,the neurotic element in the patient's constitution being largely operative.The mobile temperature of these young patients,the wider expression of movement,the large preponderance of cases in girls as compared with boys,according to the Collective Investigation Statistics nearly three to one,the occurrence

of the affection chiefly in quick,emotional children and in emotional races,the agency of fright or mental excitement as an immediate exciting cause,all point to a nervous factor.The above-mentioned statistics(p.54)yield a neurotic family history in forty-six per cent.;but,as this includes fourteen per cent.of chorea itself,and such disorders as sunstroke,injury to the spine,sciatica, shingles,tubercular meningitis,alcoholism,paralysis,etc., many of which are purely accidental,and other have obviously no connection with instability of the nervous system,they are of little practical value.Inherited neurosis is seen only in small proportion of other published cases.The presence of the neurotic or emotional factor does not exclude that of a rheumatic factor. There would seem to be a close association of the two in intimate operation,though some writers affirm that they are mutually antagonistic or destructive,only one or other of the pair being present.In proof of the intimate relation of chorea to rheumatism it may be mentioned that it is constantly seen as a sequel or an accompaniment of rheumatic fever,and of no other acute affection except scarlatina,with which rheumatism seems to have a strange relationship.At varying intervals it is followed by acute articular rheumatism in certain instances.It occurs in connection with simple articular pains,which are probably rheumatic,since exactly similar articular pains without swelling are often associated with endocarditis and pericarditis.It occurs also in conjunction with endocarditis and pericarditis,together with certain other affections of a rheumatic nature,such as subcutaneous nodules and erythema,or with these alone. When it occurs with cardiac disease without any other rheumatic manifestation,the morbid changes are mitral valvulitis and pericarditis,that is,that seen in ordinary rheumatic fever.Further evidence of our contention is seen in the association of emotional excitability with both chorea and rheumatism.The greater frequency of chorea in girls corresponds with the greater frequency of acute rheumatism in girls and the prevalence of mitral stenosis in young women.A host of statistics can also be adduced,though we may place on one side those which deal solely with rheumatic arthritis which is either antecedent to the chorea or its immediate accompaniment.For any calculation based on this point of contact alone must obviouslybe inadequate,since they take no account of the cases in which chorea comes after articular inflammation.These statistics,moreover,as a rule,deal only with marked attacks of articular rheumatism;they omit also to weigh the presumptive evidence afforded by the coexistence of endocarditis and pericarditis,of subcutaneous nodules,of erythema,and of inheritance.The statistics of the Collective Investigation Committee are of greater value.They are based upon four hundred and thirty-nine cases,and give about twenty-four per cent.(we shall disregard fraction)of antecedent rheumatic arthritis and twelve per cent.of concurrent or immediately subsequent arthritis,making a total of nearly thirty-seven per cent.unquestionably rheumatic.If five per cent.of cases with vague pains probably rheumatic were included,the proportion would be about forty-two per cent.Obviously this is an underestimation of actual facts,as the cases in which arthritis occurs some time later are necessarily excluded;

and the evidence afforded by mitral disease, by pericarditis, by fibrous nodules, by erythema, by tonsillitis, apart from arthritis, is not estimated. When these occur in combination, their cumulative weight as evidence of rheumatic connective is considerable. Barlow (loc.cit.) found satisfactory evidence of rheumatism, exclusive of family history, in fifty-seven per cent. Other statistics, taken with minute care with special regard to this point, and including in several instances many years of the after-history, give over seventy per cent. From this, however, some reduction must be made, probably of twenty to twenty-five per cent. of those cases in which family history is the only evidence, on account of the normal incidence. This would bring the estimate into close equality with that of the observer just mentioned. A careful inquiry into the life-history of the patient alone can determine whether or not a given case of chorea is rheumatic. The affection may be seen at any time during the occurrence of the rheumatic phenomena, it being a very dangerous complication when severe and combined with very acute endo- or pericarditis.

An association of erythema with rheumatism has often been affirmed, the most common form of the former affection being erythema marginatum and urticaria. The former is a common accompaniment of articular rheumatism in children, being far more often seen in them than in adults, appearing on the body as well as upon the limbs. Barlow (Brit. Med. Jour., Sept. 15, 1883, p. 313) gives a series of remarkable cases in which the marginate or urticarious form appeared simultaneously with pericarditis, or immediately preceded it, the articular affection following later, though in some of the cases it is possible that the erythema was due to the toxic action of the arsenic, quinine, or salicylate or soda administered for the relief of the symptoms. Warner and Barlow (Trans. Internat. Med. Congr., 1881, Vol. iv, p. 118) state that out of twenty-seven cases of fibrous nodules, erythema papulatum or marginatum appeared in eight. Stephen Mackenzie writes in favour of a connection between erythema nodosum and rheumatism, and furnishes statistics in illustration of his views. He records one hundred and eight cases, in sixty-seven of which arthritis occurred, and, what is significant, in two instances endocarditis developed with the eruption, but without any articular affection. Garrod reports twenty-seven consecutive cases; there was a family history of rheumatism in six and eleven suffered from arthritis. Still the claims that erythema nodosum should be included in the rheumatic series is by some not allowed. The affection is doubtless usually accompanied with pain and tenderness of joints, closely resembling a rheumatic arthritis, and in joints removed from the site of eruption, so that the pain cannot always be due to the presence of swelling on unyielding parts. Objectors further insist that until this form of erythema is unquestionably found associated with heart disease or other attacks of undoubted rheumatism it cannot be definitely accepted as of rheumatic origination. We may hesitate less in accepting purpuric erythema as a rheumatic expression. This affection, the peliosus rheumatica of Schönlein, is said to occur almost exclusively in young adults, though there have since this affirmation been several cases recorded of the malady in young children. It is to be distinguished from the

purpuric eruptions which occur in Bright's disease, pyaemia, and other forms of septicaemia, in heart disease, and in the wasting of organic affections. The special characteristic of this form of erythema indicate that the subcutaneous haemorrhages which form such a conspicuous feature of the eruption are probably due to thrombosis of the small vessels, as in septicaemias; and this is entirely consistent with the rheumatic condition, as in the latter the blood contains an excess of fibrin, and thromboses in even large veins occur during life, and after death abnormal coagulations. The malady may make its appearance as an isolated phenomenon apart from other symptoms, as has been not infrequently observed. Haemorrhage from the bladder may also occur.

Certain rheumatic cases in children may show a tendency to the occurrence of thrombosis and embolism, the existence of hyperinosis and proclivity to clot-formation being dangers sometimes obtaining. It would seem that plugging of any large vessel does not occur except when the circulation is interfered with and slowed by the concurrent mitral disease or pericarditis.

Children suffering from rheumatic fever are usually markedly anaemic. The condition is generally present in some degree in ordinary cases, but it is a remarkable and a prominent symptom when there is serious cardiac disease either valvular or pericardial. It sometimes progresses to an extreme degree, and is then a dangerous condition. Children with serious heart-damage from endocarditis or pericarditis suffer in nutrition in a remarkable manner; they not only grow strikingly pale, but lose flesh and strength, and ultimately they die from progressive weakness and failure of the cardiac action.

Nervous excitability, usually of an emotional character, is not infrequently observed; but is doubtful whether this is a direct consequence of rheumatism, or only a part of the chronic condition. The general opinion, however, seems to be that the emotional, neurotic disposition usually is associated with a tendency to arthritis. Apart from chorea, rheumatic children are abnormally excitable; they are restless, fidgety, and nervous, and difficult to deal with. Several cases have been published by way of demonstrating the alleged occurrence of meningitis in rheumatism. It is possible that the serous membrane of the brain may be excited to inflammation by the rheumatic poison, like other serous membranes. The cerebral symptoms observed in some cases, which Trousseau ascribed to cerebral rheumatism, appear to be effects of pyrexia or pericarditis, and sometimes perhaps of the exhibition of the salicylates. Nocturnal pyrexia, headache, and incontinence of urine are stated by Goodhardt to be especially common inrheumatic children. I have never observed any special relation of these condition to rheumatism, but the association with nervous excitability and anaemia would seem to establish the probability.

During the course of the rheumatic attack there may be seen subcutaneous fibrous nodules, as was first pointed out by Barlow and Warner (Trans. Internat. Med. Congr., 1881, Vol. iv, p. 116) as being of great frequency and pathological importance. Their existence had been previously noted by Hillier and other observers abroad, who, however, did not consider them of much importance. They are extremely common in children, but are rare in

adults,although cases of their occurrence in grown-up individuals have been noted by Stephen Mackenzie,Duckworth(Proc.Clin.Soc.,1883,xv),and others.These subcutaneous tendinous nodules vary in size from that of a pinhead to an almond,or even larger.They are not tender. They are found chiefly in the neighbourhood of joints, especially at the back of the elbow,about the margin of the patella,and the malleoli.They occur also about the vertebral spines,the spine of the iliac crest,along the clavicle,the extensor tendons of the hand and of the foot,the pinna of the ear,the temporal ridge,the superior curved line of the occiput,and the forehead.They have also been noted on the flexor tendons of the palms, where they may attain the size of an almond,and temporarily prevent the proper use of the hands.As many as thirty or forty have been seen,mainly confined to the front of the chest,and in relation to the tendons and fasciae of the intercostal muscles.They sometimes appear in successive crops,sometimes singly,sometimes multiple. They have been known to develop in the course of ten days;but they usually take many weeks to subside.There can be no question of their relation to rheumatism,it being the rarest thing possible for them to occur apart from that disease.In Barlow and Warner's cases there was distinct evidence of rheumatism in twenty-five out of twenty-seven;in several cases also there was chorea, and frequently marginate erythema.Their chief association,however,is with endo- or pericarditis;and this gives them an especial significance and value.They seem to make their appearance at the same time as the inflammation of the endocardium,and they are important phenomena when there is an abundant and recurrent efflorescence. They have been observed not only in connection with tendons and fasciae,but als in the periosteum in a few cases,and,according to Money(Lancet,1886,Vol.ii,p.158), in the pericardium itself;as many as two hundred have been seen on the body at one time.At one time it was though that their connection with rheumatic fever was absolute;but this connection must be expanded to include arthritis deformans,in which they also occur.Instances have been reported of rheumatoid arthritis in which, during the more active stage of pain and tenderness and swelling,a crop of typical nodules made its appearance. Some of these cases were in adults,and,contrary to the rule in children,were distinctly tender.Duckworth(Trans. Clin.Soc.,Vol.xvi,p.52),Pitt(Ibid.,Vol.xxvii,p.54), Bannatyne(Rheumatoid Arthritis,p.113),Futcher(Johns Hopk.Hosp.Bull.,1895),and other also report cases;but such,however,are rare,and most rare in children.These nodules are seldom seen in rheumatoid arthritis because they usually occur in children who only rarely duffer from arthritis deformans.

The liability of rheumatic children to <u>tonsillitis</u> is well known;it may usher in the rheumatic attack or make its appearance during the evolution of the disease. Trousseau has described a rheumatic sore-throat.The statistics of the Collective Investigation Committe(Vol. 14,p.71,1888)show that inflammation of the tonsil occurred as an antecedent to rheumatic fever in some twenty-four per cent.of cases,with ten per cent.of angina of uncertain character.This only gives instances in which tonsillitis came first in the rheumatic series;and its full significance is only realised when we consider that the throat affection occurs also as a later as

well as an initial affection,although not so frequently, and that it occurs apart from articular symptoms in rheumatic subjects.There are several cases on record in which the tonsillitis followed immediately after articular rheumatism,endocarditis,and chorea,and others in which it followed rheumatic fever and endocarditis,coming in each instance last in the series.In still other cases repeated attacks of tonsillitis extended over several years and followed an attack of rheumatic fever which never recurred,but which was succeeded by chorea and purpuric erythema.The affection would therefore seem closely associated with the rheumatic condition.It presents no special features,is accompanied by sharp fever,with a temperature of 102. to 103.F.,rarely terminates in suppuration,and may extend to the pharynx and soft palate.We have already seen that it has been suggested that it is through the tonsils that the infecting agent of the disease gains access to the system,and that the symptoms of the rheumatic fever are the outcome of the absorption of the toxic products of the exciting germ.

Bronchitis is not very often seen in rheumatic fever occurring in children;Lebert estimates its frequency at nine per cent.

Pleurisy and pneumonia are always to be regarded as serious accidents;they are,fortunately less common than the cardiac affections.Pneumonia occurs mainly in a limited form as an accompaniment of pleurisy,in a more extensive degree in relation to,and probably dependent on,mitral disease and pericarditis,and in the embolic form also in connection with valvular disease. According to Lebert,who found the lesion in ten per cent.of his cases,pleurisy is a distinct expression of rheumatism,it being doubtful whether pneumonia can claim to be considered a certainphase of the latter disease.Like pneumonia,pleurisy is most common in the left side,and frequently is associated with pericarditis. In the latter case it may be secondary,but when it occurs alone in rheumatic subjects,or as one member of a series of rheumatic phenomena,it is probably a direct expression of rheumatism.The stitch of pleurisy is often referred erroneously to intercostal rheumatism or pleurodynia.Pain in the side should never be passed over with a hasty diagnosis of this kind,but be the subject of careful examination with the stethescope. The general symptoms of pneumonia occurring in the course of rheumatism are usually rise of temperature,to 103. or 104.F. perhaps,and somewhat accelerated respiration.There is little or no cough,no characteristic sputum,even in the case of adults:in fact,nothing to call attention specially to the state of the lungs. Consequently pneumonia is frequently only discovered on routine examination;and,as auscultation of the posterior region of the thorax is often omitted in rheumatism on account of the pain which it inflicts,the existence of inflammation of the lung is very liable to escape recognition.The physical signs differ somewhat from those of ordinary pneumonia.There is bronchial or tubular respiration,but fine crepitation is not commonly observed;in the limited embolic form this is,however, usually found.Pleurisy and pneumonia occurring as simple inflammations excited by the rheumatic virus usually resolve readily,and fluid effused as a result

of the former is reabsorbed, unless, as in some cases, it becomes purulent. But should the affection be the result of cardiac disease, the consolidation of the pulmonary tissue and the effusion into the pleural cavity persist for a considerable period of time.

Several cases have been published to instance the occurrence of appendicitis in individuals of rheumatic history and predisposition; they have usually been mild, and in those recorded by Grant (Med. Rec., Nov. 11, 1893) and Yeo (Brit. Med. Jour., June 16, 1894) the attack was accompanied by well-marked affection of the joints. In other cases the family history of both rheumatism and appendicitis has been striking. Not a few of the cases from time to time recorded have not been so definite: in some the evidence of appendicitis, and in other that of rheumatism, in some of both, being by no means above suspicion. The rheumatic nature of the affection has been insisted upon especially by those who have cured the disease by the exhibition of the salicylates. Sutherland (Lancet, Vol. ii, 1895, pp. 457-459; and Edin. Hospital Reps., 1895) has seen several cases, and speaks of the number of instances of appendicitis which have been recorded in which no adequate cause for its origin could be found. The concretion discovered in some cases he regards as identical with the calculi found in the crypts of the tonsil in chronic tonsillitis, and lays emphasis on the theory advanced by some that the appendix is a kind of abdominal tonsil. It remains for the future to determine the mooted point.

D I A G N O S I S.

IN ADULTS.

The vast majority of the cases of acute articular rheumatism are easily diagnosed on the history of the case, the course and distribution of the arthritic symptoms, characterised by mobility and instability, and the simple inflammatory nature of the lesions, combined with pyrexia and tendency to cardiac inflammations.

Occasionally, however, doubt may be entertained with regard to other affections. Secondary arthritic may develop in connection with acute infections such as scarlatina, gonorrhoea, dysentery, puerperal or other sepsis, and cerebro-spinal meningitis. In each of these cases there are present the symptoms of complicating disease, and in all severe cases there is marked tendency to joint suppuration and structural change affecting the cartilages, ligaments, etc., which have no part in ordinary rheumatism.

Pyaemia is an affection which has often been mistaken for articular rheumatism and vice versa; but articular rheumatism, unlike pyaemia, is not necessarily connected with any pre-existing condition capable of causing purulent infection of the blood or system, such as a wound, fracture, abscess, or a local inflammation of bone, periosteum, vein, pelvic organ, or a specific fever, such as smallpox, relapsing fever, typhoid fever, glanders, etc.; it does not present severe rigors, which recur at

irregular intervals and are attended with chattering of the teeth and a high temperature,104. to 105.F.,rapidly attained;its type of fever is not so intermittent or markedly remittent as that of pyaemia;its profuse sweating continues although the temperature remains febrile, but that of pyaemia coincides with the decline of the temperature;unlike pyaemia,it only very rarely produces profound constitutional disturbance of a typhoid character,and has no tendency to run a rapidly fatal course in eight to ten days or in two or three weeks;its visceral inflammations are chiefly cardiac,pleural,and pulmonary,and tend to resolve;those of pyaemia are especially pulmonary,pleural,and hepatic,although frequently cardiac also,and generally produce suppuration and destruction of tissue.Multiple subcutaneous abscesses and cutaneous blebs and pustules do not occur in rheumatic fever,and its articular affection differs in many respects from that of pyaemia;many more joints are involved;the inflammation is erratic,very rarely fixed,and generally resolves without damage to the articulation;the affected joint is usually hotter,redder, more painful,and more sensitive,and the swelling is less diffused,and its outline corresponds more accurately with that of the synovial capsule.Sometimes rheumatic fever is complicated with the phenomena of pyaemia,as when malignant endicardial inflammation occurs.

The acute and subacute inflammations occasionally observed in cerebral softening and haemorrhage,in injuries and inflammation of the spinal cord and caries of the vertebrae may be distinguished from acute and subacute articular rheumatism by the following circumstances:the existence of some one of these diseases of the brain or cord,the articular affection being usually confined to the paralysed limbs;its invasion about the time of the setting in of the late rigidity,or even still later;the absence of cardiac complications and the presence of other trophic or neuro-paralytic lesions, such as acute sloughings,rapid atrophy of the paralysed muscles,cystitis,ammoniacal urine,and so forth.

The diagnosis of acute gout involving many of the joints will be greatly aided by the history of the patient,though it must be remembered that gouty subjects may sometimes experience acute rheumatism,and,moreover, a rheumatic attack sometimes commences suddenly in the night,involving the great toe,which is the favourite seat of gout.In doubtful cases the uric-acid test may become necessary to decide the diagnosis.The habits of the patient are also suggestive.

The differentiation of rheumatic fever from rheumatoid arthritis is sometimes not easy.But the site of election of the latter is the smaller joints;it has an intractable course;it does not respond to the salicylates;there is wasting of the muscles;the action of heart,which is free from disease as a rule;and the clinical history of the case is different.

Gonorrhoeal rheumatism is more apt to be confounded with mono-articular rheumatism than with ordinary rheumatic fever.But there is a history of a gonorrhoeal attack,the heart is less liable to implication,and the salicylates have no specific action;furthermore,iritis is more common than in rheumatism,the joint affection is very stubborn in its course,and the plantar fasciae are not infrequently involved.

In influenza the occurrence of articular complic-

ations, angina, and cardiac phenomena may cause confusion. But the onset of the constitutional disease is more abrupt than that of rheumatic fever, and the articular complication is usually of late occurrence and comparatively slow diffusion.

It is only in the early stage of acute glanders that the severe muscular and articular pains sometimes present in that very rare disease in man might lead to its being confounded with acute articular rheumatism; but the patient's occupation and history, the early and severe prostration, the absence, as a rule, of redness and swelling around the painful articulations, and, in some instances, the early appearance of pustules and blebs on the skin and of abscesses in the deeper tissues, will decide the diagnosis.

Typhoid fever with articular trouble may be confounded with adynamic rheumatic cases; but the occurrence of diarrhoea, splenic hypertrophy, the rose-coloured eruption, and a positive Widal reaction, and the general personal and clinical history will suggest the real nature of the case.

Malta fever sometimes resembles rheumatism; but there is a specific microbe and also a definte serum reaction therewith; it occurs with sudden pyrexia and severe prostration, the same alternating with periods of comparative health. It also prevails only in certain localities.

A sprain is sometimes put down to rheumatism; but the fact that there is a history of trauma should prevent this error beingmade.

In haemophilia, purpura, and scurvy, etc., effusions of blood with swelling and pain in the joints may resemble rheumatism; but these haemorrhagic disease have all definite features apart from rheumatism. There is usually haemorrhage elsewhere than in the joints, and fever, if present, is not intense.

IN CHILDHOOD.

In children the typical course of rheumatic fever in the adult is seldom observed, so that the diagnosis of the disease in these young patients, when arthritis is at its minimum and fever and perspiration are not pronounced, is often by no means easy; and the nature of the case can then only be decided by the careful study of the illness and other circumstances obtaining. Characteristic features are inflammation of the joints, the articulations being swollen and tender and painful, with a faint blush of redness on them perhaps, the tendency to sweating, the rise of temperature to the height already named, the behaviour of the inflammation in shifting from one joint to another, and declining and reappearing in a few days. When the tenderness and swelling are extremely slight, confined to a single joint, or in a tendon or fascia, or if there is merely a little stiffness, it is occasionally difficult to decide whether the inflammation is really rheumatic or not, although the mere existence of anything of the kind in the case of a child is in itself very suggestive of rheumatism. Such conditions are usually rheumatic, and are, it is to be remembered, genuine rheumatism, bearing with it all the possibilities of cardiac inflammation. How constantly these cases must be overlooked is shown by the fact that out of six hundred and fifty-five cases of all ages given in the statistics of the

Collective Investigation Committee(loc.cit.) thirty-two only were in children under ten;yet,as already stated,it is one of the commonest diseases encountered in practices,hospital or private,dealing considerably with the affections of childhood.The discovery of a fibrous nodule,or the rash of erythema,or a mitral murmur,or pericardial or pleuritic friction,or that the patient has had a previous attack of rheumatism in any form,or inherits a family taint of it,will help to determine the diagnosis.All these points should be minutely inquired into,and in every case the heart carefully examined day by day.

There are,however,one or two affections which might be mistaken.One of these is acute osteomyelitis, which in its early stage presents certain points of resemblance to acute rheumatism.There are pain and tenderness of limb,accompanied by fever,and the disease occurs inchildren more frequently perhaps than in adults.It is distinguished by the seat of the pain and tenderness being not in the joint,but in the shaft of the bone(usually one of the long bones,near its end, and close to the epiphysis),and by the extreme intensity of the pain and tenderness,by the swelling of the periosteum,and by the great severity of the constitutional disturbance.The pulse is rapid,and the temperature is high,and the general features of the case are out of all proportion to the local trouble.

Acute periostitis frequently occurs in children in close proximity either to one joint,or less frequently to more than one,and may readily be confounded with acute articular rheumatism.But the constitutional disturbance in acute periostitis is prompt and severe at the outset;the swelling increases rapidly,is firmer than that of arthritis,does not involve the joint proper and its capsule,but,ilke the tenderness on pressure, exists above or below the articulations,especially round the head of the bone;there are no visceral complications,provided pyaemia has not supervened;the constitutional symptoms early assume a typhoid character, and the patient soon dies in the absence of a surgical operation.

The enlarged ends of the long bones and the pains in the limbs of rickets may lead to a suspicion of acute articular rheumatism,but the early age of such children,the absence of pain and swelling in the joints, the beaded condition of the sternal ends of the ribs, the late dentition and locomotion,the peculiarly shaped head,and other evidences of that affection,would prevent a careful observer from making a mistake.

Inhetited syphilis in infants,like rickets,may produce fusiform sweling and thickening of the ends of the long bones,especially the humerus and femur,and sometimes pain in the joints on movement;but at first the swelling is confined to the epiphyseal line,and only later extends to the joint;there is a pseudo-paralysis of the limb,and but little pain or fever;bony osteophytes may be often felt under the skin at the line of union of the epiphysis with the shaft;the epiphysis often becomes separated from the shaft,and suppuration may ensue around the bone and in the articulation;sometimes adhesions and perforation of the integument takes place,allowing of the escape of disintegrated osseous and cartilaginous tissue;and there

will coexist some of the ordinary manifestations of congenital syphilis - Hutchinson's teeth, snuffles, and so forth.

Owing to its frequent early accompaniments of pain and pyrexia, poliomyleitis anterior may be mistaken for rheumatism. But there is loss of reflex and wasting; and heart disease is not observed in uncomplicated cases.

Infantile scurvy is occasionally mistaken for acute rheumatism; but the former is common under two years of age, when the latter is very exceptionally seen. In scurvy there are ecchymoses on the gums, periosteal swellings, purpuric eruptions, haemorrhages from the kidneys or mucous membranes, the face is extremely pale, the heart is not involved, and dieting of the patient gives signal relief.

Gonorrhoea arthritis is sometimes the outcome of infection from the mother; it shows itself during the first five weeks of life, is associated with inflammation of the eyes, and fails to respond to the salicylates.

Appendicitis has sometimes caused confusion in cases in which the rheumatic process is located in the right hip-joint, the patient referring the pain to Mc Burney's point above Poupart's ligament. But the careful study of the history and symptoms should solve the problem and save the patient from a useless operation.

Acute multiple tuberculous arthritis is almost impossible to distinguish from sometimes from acute articular rheumatism. But the tubercle bacillus can usually be isolated from the lesion, pyrexia of rheumatic type is absent, the history of the case is different, and tuberculous conditions are generally observed elsewhere.

Arthritis deformans is seldom seen in childhood, but when it does occur may cause difficulty in diagnosis. The general and personal history of the case must here be relied upon for solve the problem.

P?R O G N O S I S.

IN ADULTS.

Rheumatic fever is one of those diseases in which to prophesy is often to meet with failure. Generally, however, the disease is seldom fatal during the attack, yet as the frequency of the complications varies unaccountably from time to time, so the mortality may be exceptionally large or small for even prolonged periods. It may be stated that the average death-rate ranges from one to four per cent. in the experience of modern authors. The average mortality in the Parisian hospitals, during a period of four years, Besnier (Dict. Ency., S.3, T.iv, p.463) fixes at nearly two per cent; in St. Bartholomew's Hospital, Southey found it for fifteen years to be a trifle over one per cent.; Pye-Smith (loc.cit.) fixes the rate at four per ent. in four hundred cases treated at Guy's; Carter gives two and a half per cent. as the rate during ten years at the Southern and Royal Hospitals at Liverpool (Liverp. Med.-Chir. Jour., 1881, p.88). The death-rate appears to vary remarkably with age, as

Southey's(loc.cit.)figures show:under ten years,3.40 per cent.,;between ten and fifteen,1.5 per cent.;between fifteen and twenty-five,1.4 per cent.;between twenty-five and thirty-five,0.9 per cent.;between thirty-five and forty-five,0.8 per cent.,the mortality declining very rapidly after the tenth,after the twenty-fifth,and after the forty-fifth year of life.The danger to life is usually proportionate to the youth of the patient, the degree of the fever,the number of the joints involved,and the number and character of the complications, the habits,and the previous health of the patient. A fatal issue is most frequently observed in connection with hyperpyrexia alone,or in combination with delirium and coma.A rapid rise of temperature and a temperature over 105.F.,especially if cerebral disturbance exist as well,indicate danger;and so does arrested perspiration when the temperature is high.In a much smaller number of cases death is due to some other complication, especially to purulent pericarditis or to that combined with pleurisy and pneumonia;in not a few cases the prior existence of chronic valvular disease,with fibroid induration of the liver and kidneys,renders a fresh rheumatic endocarditis or pericarditis,occurring as a part of acute articular rheumatism,fatal.There is good,if not conclusive,evidence that rather sudden death in acute articular rheumatism is occasionally due either to diffuse myocarditis or fatty degeneration of the heart.In Greenhow's two deaths out of fifty cases treated by sodium salicylate the pericardium was universally adherent and the heart's fibre fatty in one and pale and flabby in the other.Sudden death in this disease is very rarely due to embolism of the pulmonary artery or of the cerebral vessels,while ulcerative endocarditis is very exceptionally one of the cources of a fatal issue.But though acute articular rheumatism seldom kills directly,it frequently lays the foundation of subsequent ill-health,and ultimately proves fatal through organic disease of the heart and its many consequences.However,it is an interesting circumstance that while acute articular inflammation is prone to damage the heart permanently,it very rarely,quite exceptionally,impairs the structure or functions of the joints.It is almost solely the subacute form that now and then becomes chronic or renders a joint for a long time painful,swollen,and crippled in its movements. Whether acute rheumatism,however intense per se,ever ends in destructive suppuration and ulceration of a joint is doubted by some authorities,notwithstanding the cases published by Fuller and others.Doubtless some of the cases were really pyaemic,or perhaps gonorrhoeal;and it must be borne in mind that acute articular rheumatism occasionally develops pyaemia,and then an arthritis might be considered rheumatic when truly pyaemic.

The prognosis in rheumatic cardiac cases will depend on the nature and intensity of the heart affection,as well as of the parent rheumatic attack.This is especially true as regards pericarditis,which is also influenced by the age at which it occurs.It is very fatal in children and the aged.A guarded prognosis should be given until it can be determined whether or not the effusion will become purulent.A low grade of circumscribed pericardial inflammation tends of

itself to recovery,but if the inflammatory action be diffused and of a severe type,the prognosis is grave. Sudden and intense pericarditis usually terminates fatally.The rapidity with which the effusion takes place influences the prognosis to a greater extent than the amount of the liquid.Sudden death may occur in a few hours from the onset of the disease from compression and paralysis of the heart.Pericarditis complicating rheumatic fever ends in recovery in the majority of instances.The prognosis is unfavourable when hyperpyrexia attends its development.The development of marked nervous manifestations does not of necessity add to the gravity of the prognosis;the latter is unfavourable in cases where there is reason to believe that myocarditis accompanies the pericarditis.In rendering a prognosis the character of the exudation must be taken into consideration.Death usually supervenes when it is haemorrhagic or purulent.The success,however,attending operative interference in purulent cases renders the prognosis more favourable than formerly.Pus is rarely absorbed. Occasionally acute pericarditis passes into the chronic form,or,rather,is accompanied by a large effusion which disappears slowly.Relapses are likely to occur,and thus the disease drags on for months.As a result of the long-continuance of the effusion the heart muscle undergoes extensive degeneration,its propelling power is diminished,and dilatation results.During the progress of the disease the patient suffers from repeated attacks of extreme dyspnoea,and death may take place from sudden syncope or oedema of the lungs.Any sudden effort may cause instant death.If recovery follows dilatation of the heart,compensatory hypertrophy is developed.When adhesions form the prognosis will vary according to their extent and situation.Circumscribed adhesions may give rise to no symptoms.Extensive adhesion of the pericardial surfaces is followed by dilatation,and the heart wall is left permanently weakened.Adhesions about the base are likely to interfere with the coronary circulation and lead to fibrosis or atrophy of the heart.

The vast majority of the cases of simple acute endocarditis terminate in valvular disease of a fibrotic character.Complete recovery in rheumatic subjects is rarely encountered,for the tendency to recurring endocarditis is very great,and there is always more or less thickening and induration of the valves remaining after the primary attack.When extensive myocariditis and pericarditis coexist,or where the rheumatic attack has produced a great depression,endocarditis must always be looked upon as a dangerous condition.The tendency to relapsing endocarditis is sometimes marked in rheumatic cases,and in such the constant reappearance of inflammatory lesions so prolongs the case and depresses vitality that the outlook for final recovery becomes a gloomy one,and the establishment of chronic valvular disease, in case of recovery,almost certain.Embolic manifestations,gastro-intestinal disturbances,pneumonia,and other complications necessarily increase the liability to fatal termination.The duration of the disease is always uncertain,and the recurring disease is,as stated, especially to be feared.

A complication of rheumatic fever with malignant endocarditis spells certain death - at least that is my firm opinion.

The prognosis in cases of valvular disease of the heart must be based upon a careful study of the nature of the disease and the degree of compensation present, as well as upon the general health of the patient, his occupation, and his habits. Provided full compensation is maintained, although the lesion be a serious one, the immediate outlook is not unfavourable. Any statements as to duration of life in valvular diseases and their relative frequency as causes of death are unreliable. Each case must be judged on its own merits. Age is an important factor in the prognosis of any case of valvular disease. The outlook in very young children is always unfavourable, for the reason that the valve lesions are apt to be progressive on account of the liability to recurrence of the rheumatic attacks. Valvular lesions which make their appearance in early adult life are more likely to be permanently compensated than those which develop in middle life or in children. The prognosis in valvular diseases which develop in middle life in those who give evidence of extensive arterial changes if always unfavourable. In old age extensive valvular insufficiencies are well borne and give rise to few urgent symptoms on the part of the heart. Females bear valvular lesions better than men, except during the childbearing period, for arterial changes are less likely to occur in them and they lead quieter lives. After the establishment of complete compensation the prospect of its continuance will be largely influenced by the patient's occupation and habits. If he be required to continue at active and laborious work and be exposed to vicissitudes of weather, the liability of early failure of the cardiac power is very great. Similarly, indiscretion in diet, the immoderate use of alcoholic stimulants and tobacco, or undue mental excitement will constantly tax the strength of the heart, and eventually aid in destroying compensation. It is by no means implied that moderate, healthful exercise of body and mind are to be looked upon as dangerous, for many patients undoubtedly are thereby benefited, and are enabled to continue long and useful lives in spite of a serious defect of one of the valves of the heart. Intercurrent diseases of all kinds render the prognosis grave. The rapid supervention of cardiac degeneration and dilatation in the course of severe illnesses is well known, and suggests special precautions to be taken in valvular persons taking rheumatic fever. First of the valvular lesions in order of gravity stands tricuspid incompetency; second, aortic incompetency; third, mitral stenosis and incompetency; fourth aortic stenosis; and fifth, stenosis of the pulmonary orifice. Tricuspid regurgitation is generally itself the result of beginning failure of compensation in a case of mitral disease, and therefore the prognosis is particularly grave. Tricupsid stenosis, on the other hand, may permit comparatively comfortable life for many years. Among the forty-six cases analysed by Fenwick, the duration of life was from thirty-one to thirty-six years. Aortic incompetency is a very serious form of valvular disease. It is impossible to estimate the probable duration of life with any certainty, for the coronary arteries may not for a long time become sufficiently involved to render the case a serious one. Sooner or later, however, they will undergo changes which will cause the outlook to become serious. It must always be remembered that when when fibrotic changes take

place at the origin of the aorta the coronary arteries are liable to undergo similar changes, and under such conditions sudden death is possible from embolism of one of their branches. In ordinary rheumatic cases aortitis and obstructive changes in the coronary arteries are of rare occurrence; consequently the prognosis in such aortic insufficiencies is comparatively good, except in young children. When in aortic insufficiency cyanosis and dropsy result from the failure of a dilated and hypertrophied left ventricle to empty itself, the prognosis is unfavourable. Acute ventricular dilatation produced during violent or prolonged physical exertion in one who has extensive aortic insufficiency is rarely recovered from, and larks the commencement of a period which soon terminates in fatal cardiac insufficiency. When the hypertrophy and dilatation are moderate in degree, and there are no urgent cardiac symptoms and no signs of extensive arterial changes, aortic regurgitation may be fully compensated for years; but if it is complicated with mitral disease, with a dilated feeble ventricle and extensive arterial changes, the prognosis becomes most unfavourable. The prognosis in aortic stenosis is less unfavourable than in any other valvular lesion. Life may be prolonged and good health enjoyed for many years if the ventricular hypertrophy fully compensates for the obstruction; but as soon as the compensatory hypertrophy fails to overcome the obstruction and dilatation begins, the action of the heart becomes feeble and intermitting. The patient is subject to attacks of vertigo and syncope; there are great muscular prostration, marked pallor of the face, with marked anginal symptoms after or during excitement. In such cases sudden death may occur by a complete arrest of the ventricular systole. Mitral stenosis admits of but slight compensation. If extensive, it is always a serious disease. The prognosis must be determined in every case by the severity of the thoracic symptoms. When during active exercise the lungs become congested and oedematous, with pulmonary haemorrhages, extreme dyspnoea, and cyanosis, the prognosis is especially unfavourable; sudden death, judging from published statistics, would seem to be as frequentl in mitral stenosis as in aortic regurgitation. Mitral incompetency is more compensated for than any other valvular lesion. The changes which lead to its development occur slowly, and their tendency is to remain stationary except in young children. Patients with moderate insufficiency suffer very little except during or after violent exercise, and were it not for the slight dizziness whichoften follows such exertion it would pass unnoticed. In all cases where the compensation is perfect it is unnecessary to tell the patient that he has heart disease, for in no other valvular lesion is he so likely to live to old age without suffering any inconvenience. When, however, the compensatory hypertrophy of the right heart gives place to dilatation and fails to overcome the obstruction in the pulmonary circulation, dyspnoea, cyanosis, with disturbances of the systemic circulation and oedema of the extremities, mark the beginning of the end. One may regard as a favourable sign freedom from pulmonary congestion or any marked disturbance in the hepatic and renal circulations after prolonged physical exertion or excessive fatigue in one who suffers from mitral regurgitation.

Sudden death sometimes occurs in cases of acute

dilatation of the heart;even when death does not immediately follow in all cases,it may ensue from failure of compensatory hypertrophy to occur.Without estimation of the extent of the dilatation it is impossible to give a prognosis.This can be done best by observing the effects of complete rest in bed.If the heart regains its control over the circulation within a short period and cardiac symptoms do not return upon moderate exertion, a favourable prognosis may be given.If,on the ontrary, the circulatory disturbances persist,the prognosis is unfavourable as regards the heart itself,though not necessarily unfavourable as regards the duration of life.Under appropriate treatment the heart may be able to perform its work until the balance of the circulation is restored by hypertrophy of its walls.But it is probable that the heart will always remain in a weakened condition in these cases,and be unable to sustain undue demands on its potentiality.

Slight diffuse myocarditis is usually completely recovered from,but the severer grades may lead to a fatal termination in a few days or a week.Recovery may ensue even in the suppurative form,as the calcareous remnants in the cardiac walls would indicate.Yet death is by no means infrequently observed.The occurrence of chronic myocarditis is always grave.So long as diuresis can be maintained the case continues fairly well.

IN CHILDREN.

In childhood a first attack of rheumatic fever is seldom fatal;and the prognosis in early life is therefore usually favourable.The mortality is about three and a half percent.The affection is,on the whole,less dangerous than in adults,the latter being exposed to the risk and dangers of hyperpyrexia.Now and then children die in a first attack of rheumatic fever from thrombosis and embolism.As a rule,however,in a primary seizure the articular symptoms are recovered from,the endocarditis or pericarditis subsides,and if there be tonsillitis or erythema they seldom persist many days. Should chorea be present,it may libger on and relapse, but generally it vanishes in the course of a few weeks from its appearance.If the primary attack rapidly ameliorates,if at most only a soft cardiac murmur at the base of haemic character has been developed,or even of a mitral murmur has appeared and subsided,it may be expected that little damage has been done.But even then it is necessary to give a guarded prognosis.It is only by careful examination for long afterwards that the extent of cardiac mischief can be safely estimated.If a mitral or aortic murmur does not die away,or reappears and grows distinct,and signs of dilatation and hypertrophy follow,there is certainly a serious valvular lesion present.Owing to the greater liability to endocarditis and pericarditis in early life,the risk of permanent damage to the heart structures is greater also. Compensation is no doubt more perfect of the lesion is small,and more quicly attained with children than with adults;but,on the other hand,if the valvular injury is great,the subsequent changes of hypertrophy and perhaps of dilatation also progress more rapidly,owing to the more ready growth of tissues in the young and their more yielding character.It must also be remembered that danger is not over withthe subsidence of the primary attack;it lies partly in the after-effects of the

cardiac affection, and less in the severity of the primary attack than in the frequent immediate relapses or the return of the disease at longer intervals. In children the cardiac inflammation has a tendency to go on smouldering in subacute form, and thus by chronic changes or frequent repetition to produce a grave cumulative result. If there have been previous attacks of rheumatism, and especially if the valves of the heart have been already injured, the prognosis is much less favourable than in the first attack. The cardiac inflammation is apt to revive, and the damage to the valves or pericardium to increase. The worst cases are those in which the heart already shows signs of great enlargement, evidenced by heaving, diffused impulse, and increased area of cardiac dulness. Fresh endocarditis and pericarditis are then especially apt to supervene, often accompanied by pleurisy; dyspnoea and rapid action of the heart follow, there is progressive anaemia, and the patient sinks from failure of the embarrassed and weakened heart. Probably in such cases there is myocarditis. Pleurisy and pneumonia are also apt to occur; they are chiefly dependent upon the cardiac lesion and consequent impediment to the circulation, and this, of course, adds to the gravity of the prognosis. Vomiting is an unfavourable sign when it occurs persustently in the course of pericarditis. Marked anaemia is a sign of evil omen: it is one of the most striking results of cardiac rheumatism in children. It is as characteristic of mitral disease and pericarditis of children as of aortic insufficiency of grown-up individuals. The congested face so frequently seen in patients with mitral disease is rarely encountered in children; they are always pallid and bloodless-looking, and instead of being well-nourished they are wasted. Dropsy is rarely seen in these rheumatic heart cases of children, who seldom exhibit the enormous swellings and waterlogging, from dilatation of the heart heart due to mitral disease, of adults. In theopinion of some writers the copious and repeated evolution of fibrous nodules is regarded as of evil omen. They go especially with chronic valvulardisease and pericarditis; and so long as the nodules continue to appear there seems reason to fear progressive heart mischief from a similar subacute inflammatory process in the fibrous tissue of the valves and pericardium. Such cases do badly, as a rule; they have constant and repeated relapses, progressive heart mischief, and progressive anaemia, and not infrequently they prove rapidly fatal in the course of a few months. Another danger is copious effusion into the pericardium; this can be estimated by the increasing dulness, muffling of the heart sounds, and upward displacement of the apex beat. Nevertheless this is far less common than in adults; an adhesive pericarsitis with subsequent fibrosis is the rule with children, rather than one with copious effusion. A less common sign of the greatest gravity is supervention of dropsy, indicating extreme dilatation of the right heart. The prognosis is also rendered very unfavourable by the supervention of severe chorea, that is, a chorea so severe that the patient is scarcely able to be kept in bed or obtain sleep on account of the violence of the jactitations. The cases recorded in the literature do not seem reliable, since they make antecedent arthritis the sole test of rheumatism; yet these appear to show a special

relation to exist between fatal chorea,rheumatism,and the age of puberty in girls(Stuges,- Chorea,pp.77-80). The coexistence of endocarditis or of old valvular disease,and still more of pericarditis,especially if accompanied by signs of effusion,with embarrassed,quick, irregular action of the heart,and dyspnoea,or of very active plastic inflammation,renders the condition more dangerous still.In such cases the prognosis must always be serious and doubtful;and the expectation of favourable issue will depend chiefly upon the decline of the cardiac symptoms,the ability to take nourishment,and to sleep.The occurrence of thrombosis is dangerous,of course.Repeated attacks of articular inflammation may give rise to permanent alterations in one or more joints,and culminate in thickening and ankylosis,though this does not often occur.Fatal pyaemia may result from suppurative rheumatic conditions.

T R E A T M E N T.

PROPHYLAXIS.

Individuals who have strong rheumatic inheritance or who have had rheumatic symptoms should especially protect themselves from cold,excessive muscular fatigue, and exposure.They should wear Jaeger woollen garments next their skin to absorb perspiration in winter,or the warm ninen-mesh garments which admit evaporation of the perspiration as it forms.The advice given by Eisenmann (Die Krankheitsfamille Rheuma,Erlangen,1841)for avoiding the affections caused by chill should be laid to heart,viz.,to continue the exercise by which the body was heated just before the exposure to cold or wet,and to defer repose until a complete change of garments has been effected.This advice is usually followed,on the promptings of personal experience,by those who are much exposed to the weather while engaged in active exercise.Another precuation is active friction of the skin.It is assuredly impossible to prove that in any given case a chill-disease,and especially an attack of rheumatism,has been prevented by one or other of the measures just alluded to.But the same uncertainty clings to all our methods of prevention.We do know, however,that the operation of those measures is diametrically opposed to certain of the consequences due to chill.Continued muscular exercise,and the development of heat which it entails,supply the losses due to refrigeration of the surface,and prompts the flow of blood to the periphery,that turgescence of the skin and muscles,which the chilling process tends to arrest. Friction of the skin has the same effect,and serves, moreover,to stimulate the cutaneous nerves.It would be as well for persons predisposed to rheumatism to abstain from overindulgence in sweets,and especially from liquors of all kinds,and to avoid all conditions producing constipation and functional inactivity of the liver.The occurrence of uric acid or calcium oxalate

in the urine in excess should be the signal for a temporary reduction in animal food and the drinking of more fluid.The skin should be kept in good condition by daily cold bathing,followed by vigorous friction,and outdoor exercise should be enjoined.All possible should be done to promote the proper housing of the poor,on whom the incidence of the disease is undoubtedly very great,and the prognosis of a rheumatic attack in a cold,damp dwelling is rendered more unfavourable than it would be under happier conditions of housing.Houses on cold clay soil should be avoided,and changeability of the weather should call for the exercise of special personal precautions on the lines laid down.

MANAGEMENT OF THE ACUTE ATTACK.

Forthwith on the commencement of an attack of acute articular rheumatism,the patient should be put to bed on a smooth and comfortable mattress,which is covered with blankets,and a careful nurse shouldbe secured, having sufficient strength to lift the patient when necessary and save him all voluntary motion.If the patient is willing to lie between blankets they are much better than sheets.Throughout the illness a bedpan and duct should be used to save the patient unnecessary effort.The bowels should be freely opened at first by a dose of calomel,and kept open thereafter by daily morning doses of Rochelle salts or aperient water.Diet is of importance in this disease;but the statement that any diet may act as a predisposing factor is a conjectural one,for the exact relation that diet bears to the disease has not yet been proved.Only in so far as they lower the vital resistance are improper and insufficient food responsible.The management of the diet during the acute stage of the disease is similar to that of other acute fevers.Some diversity of opinion exists as to what constitutes the best diet in these cases.Some allow animal broths,and say that they have never seen any advantage result from cutting them off entirely;whereas others prescribe a somewhat more liberal diet than one is accustomed to allow in febrile affections.The safest plan during the acute attack is to put the patient on a milk and farinaceous diet.If the patient cannot take milk,milk-toast,barley or arrowroot gruel,buttermilk,kumiss,and if these are not sufficiently ample, soups and broths may be given.Meat-extracts are contraindicated,and it is well,as far as possible,to avoid animal broths.Bland or acid drinks may be given freely for the thirst,which is always,or usually,a distressing symptom.Lemonade is generally serviceable,especially since lemons have been advocated for the treatment of the disease.It is quite in order to have recourse to carbonated waters or Vichy, and milk and carbonated water,buttermilk or Kumiss for this symptom.The patient should be allowed only milk and farinaceous food until the convalescence is established,that is,for a week or ten days after the fever has subsided.The return to solid food should be gradual.Fish,oysters,and eggs should be added first,followed by chicken,and later by other meats.Vegetables may be added at the same time, the more easily digested,such as well-baked potatoes and well-cooked spinach,cauliflower tops,stewed celery, and the like, being chosen first.Sweets are to be avoided,but fresh fruit may be taken.If there be anaemia, and there usually,as we have seen,nearly always is,it

is necessary not to delay too long the return to solid food.In all possible ways the patient's strength should be fostered.His digestion should not be taxed by meals of too large a size;they may be supplemented by two or three extra glasses of milk a day,served with a piece of toast or a biscuit.In the acute stages of the disease alcohol is contraindicated,but may be prescribed for weak patients,and where cardiac complications exist. Continued weakness during the convalescence will also warrant its judicious administration.

MEDICINAL TREATMENT.

In view of the incompleteness of our knowledge regarding the real nature of rheumatic fever,its treatment is still largely empirical or intended to combat certain prominent symptoms or complications of the disease.The necessarily limited character of this dissertation will obviously prevent the complete description of the innumerable methods of treatment which have during the past been employed;even of those advocated by enthusiasts at the present time only a very few need be considered.

SALICYLATES.

The method which is undoubtedly the favourite one everywhere,and which in its power in relieving the articular and muscular pains promptly and reducing the fever of acute articular rheumatism may without exaggeration be compared to that exercised by quinine over the paroxysms of malaria,is that in which salicylic acid or the salicylate of sodium is given in repeated and full doses.It was in July,1875,that Buss first asserted (Die Antipyr.Wirkung der Salycylsäure,-Centralbl.f. Med.Wissenschr.,1875,276)that salicylic acid was a specific for rheumatism,and in March,1876,Maclagan(Lancet, March 4 & 11,1876),after having employed salicine from 1874,published his experience of it as a valuable remedy in the treatment of acute rheumatism,its beneficial action being"generally apparent within twenty-four, always within forty-eight,hours of its administration in sufficient dose".A sufficient time has now elapsed since then to permit of a just opinion of the power of these remedies,the salicyl compounds,over acute articular rheumatism;and,taking an all-round view of the question,we may conclude definitely as follows:The articular pain and the fever of rheumatic fever are more or less speedily removed by the salicyl compounds,salicylic acid,sodium salicylate,and salicine;the pains very frequently persist after the temperature has become normal.Both symptoms were removed by five days' use of such agents in fifty per cent.,and by eleven days' use in eighty per cent.,of three hundred and fifty-five cases treated at Guy's Hospital and tabulated by Fagge (Lancet,1883,ii,1031),and by five days' use in sixty per cent.,and by eleven days' use in sixty-six per cent., of the sixty severe cases treated and severely criticised by Greenhow(Trans.Clin.Soc.,1880,Vol.xiii).Again, in one hundred and ninety cases of acute and subacute articular rheumatism the average duration,under salicyl remedies,of pyrexia was,according to Warner(ibid.,p.1080), 5.5 days and of joint disease 5.3 days;in one hundred and fifty-six cases at St.George's Hospital the average duration of pyrexia was,according to Owen(ibid.,p.1081), 3.66 days,of pain four days;in eighty-two at the

Middlesex Hospital the average duration of pyrexia was five days, of pain five and a half days - Coupland (ibid., i, 1882, 10); and in fifty-five at the Westminster Hospital the average duration of pyrexia was seven days, of pain seven and a quarter days (ibid., ii, 1881, p. 1080) - that is, a general average duration in the whole series for the pain and pyrexia of about five and a half days. Moreover, thirty-six per cent. of Fagge's cases and fifty-eight of Greenhow's were relieved of both the symptoms mentioned on the fourth day; twenty-five per cent. of Fagge's and fifty per cent. of Greenhow's on the third day; and thirteen and a half per cent. of Fagge's and twenty-six and a half per cent. of Greenhow's on the second day. In Clouston's (Practitioner, 1882, i) twenty-seven cases, treated privately, sixty-six and a half per cent. were free from pain and seventy-two and a half per cent. of fever within four days. Indeed, all who have had much experience of this method of treating acute articular rheumatism will agree that the first or second dose frequently relieves the articular pains like a charm, and the local swelling then frequently subsides in from sixteen to forty-eight hours. It is said that relapses are more frequent, probably considerably more frequent, under treatment by salicylates than under other methods. Thus, the average of relapses in eight different tables of cases treated by salicyl remedies ranged from sixteen and a half per cent. to thirty-five per cent., giving a general average of about twenty-six per cent. (Fagge's 26.2 per cent.; Greenhow's, 35; Warner's, 33.6; Owen's, 30.2; Hood's, 18.8; Coupland's, 35.3; Broadbent's, 16.6; Powell's, 18.7; total, 214 ÷ 3 = 16 per cent.); while under other methods in three different tables the average ranged from 5.4 per cent. to 27.6 (this last under the full alkaline), giving a general average of sixteen per cent. (Hood's, 5.4; Warner's, 14.9; Owen's, 27.6; total, 47.9 ÷ 3 = 16 per cent.). Relapses appeared to recur less frequently in those cases which yielded under salicylates within five days than in those which took from six to eleven days to yield, in the ratio, according to Fagge's figures, of 26.6 per cent. for the first, and 29.4 per cent. for the second day; and, according to Hood's as 18.4 per cent. to 24.4 per cent. There does not appear to be any regularity in the order of occurrence of recurrence of relapses, nor is Southey's definite statement, that in "relapsing cases the temperature is nearly or quite normal on the eighth evening, and a slight relapse occurs on the thirteenth morning" borne out by my own cases. Moreover, W. Carter's (Liverp. Med.-Chir. Jour., July, 1881, p. 101) cases have not confirmed Southey's precise statement respecting the gradual remission of the temperature on the eighth and ninth days of illness in the continued or non-relapsing, uncomplicated forms. The relapses under treatment by the salicylates have been referred to the premature disuse of those remedies, but they do occur notwithstanding the continued exhibition of them. It is generally believed that exposure to cold, errors in diet, and an early return to work are frequent causes of relapse; and Boradbent refers the increased liability to relapse under salicyl compounds to the rapidity with which these remedies relieve the acute symptoms of rheumatic fever, in consequence of which sufficient care is not observed either by the patients

or their nurses,and they are exposed tosome of the above exciting causes of relapse.All the above causes do probably play their part so long as the rheumatic poison has not been wholly eliminated or destroyed.Indeed, the short intervals which frequently obtain between the primary invasion of the so-called relapses,and the failure of the salicyl compounds to prevent pericarditis or endocarditis,render it probable that what are commonly spoken of as relapses are not due to a new infection,as in the case of the relapse of typhoid fever,but to the recrudescencesof a disease not yet terminated,but over some of the manifestations of which,the articular inflammation and the fever,the salicylates exercise some control.It is generally held that the salicyl compounds do not arrest or control rheumatic inflammation of the endocardium or pericardium or pleura,or subdue the pyrexia,of these complications in well-marked degree exist; and there is strong evidence to show that they do not at all constantly prevant the disease from involving these organs,even after the articular affection has subsided under their use.Inestimable as is the benefit conferred by these remedies in promptly relieving the articular pain and fever,they do not secure the great desideratum in the treatment of rheumatic fever,protection of the heart,in every instance.In three hundred and fifty-two cases treated with salicylate of soda at the Westminster Hospital,heart disease developed in 13.6 per cent.;in two hundred and sixty-seven treated without the salicylate,heart disease developed in 14.2 per cent.(Warner,- Lancet,1881,ii,1080).In three hundred and fifty treated with salicylates at Guy's,heart complications obtained in sixty-eight per cent.,while in eight hundred and fifty treated without them,the cardiac complications occurred in 58.8 per cent.(Hood,- ibid.,1881,ii,1120).Gilbart-Smith collected a large number of cases from several of the London Hospitals, and analysed them with the following results:Of one thousand,seven hundred and twenty-seven cases of acute rheumatism treated before the introduction of the salicyl compounds,the proportion of cardiac complications was 54.4 per cent.;in seventeen hundred and forty-eight cases treated sunsequently to their introduction, the cardiac affections obtained in 63.4 per cent.;and in five hundred and thirty-three cases treated by the salicyl compounds,those affections obtained in 68.4 per cent.(ibid.,1882,i,136).These facts would seem to prove that the salicylates do not prevent the occurrence of the visceral complications or manifestations of acute articular rheumatism;and,were space unlimited,I could quote instances from many authors in which either endocarditis or pericarditis or pleurisy or pneumonia or other visceral manifestations had set in after the patient had been taking the salicylates long enough to have produced their usual physiological effects;some of these I shall presently refer to.It may be objected that in the above estimates sufficient attention has not been paid to the period of the disease at which the treatment by the salicylates was commenced,the time it was continued,the doses given,the age of the patient, the severity and other characteristics of the illness, such as whether acute or subacute,first or second attack,complicated or uncomplicated.It must be admitted that there are a few facts which render it very

probable that the salicyl compounds do really reduce the frequency of these complications, and this give some protection to the heart in rheumatism. Of Powell's (Lancet, 1882, i, 134) thirty-two cases, nineteen (equal to sixty per cent.) had heart disease when admitted; and of the remaining thirteen, six (equal to forty-six per cent.) developed cardiac disease after admission and while under the salicylates. Of Jacobi's (St. Thomas' Hosp. Reps., N.S., viii, 252) one hundred and fifty cases, seventy-eight, or fifty-two per cent., were admitted with unsound hearts, and of the other seventy-two, only five, or 6.9 per cent., developed cardiac disease after beginning the salicylate treatment. Of Southey's fifty-one cases, twenty-four, or forty-seven per cent., were admitted with diseased heart's; and of the remaining twenty-seven, only four, or 14.8 per cent., developed a cardiac affection subsequent to beginning treatment by the salicylates (St. Barthol. Hosp. Reps., xvi, 10). No heart affection was developed in any of Clouston's twenty-seven private cases - a result he attributes to the early period at which the remedies are given in private practice. But the number is too small to admit any conclusion being drawn, and four of the cases were examples of recurrence of the disease at short intervals, three or four weeks, in the same patient, in whom there appears to have existed no proclivity to cardiac complication, for he had had four attacks before he came under Clouston's case. Moreover, his cases were mild, but sixteen of them being acute, and of these only three attaining a temperature of 103. F. and upward. Furthermore, Hermann (Quoted by T. G. Smith, - Lancet, 1882, i, 137) estimates the percentage of cardiac affections that developed after beginning the salicylates in the London Hospital at 18.7 per cent., and after other treatment at thirty per cent. Omitting Clouston's, the general average of the above results is, that in 49.2 per cent. cardiac disease existed before the patients began the salicylate treatment, and that in 18.2 per cent. it developed after that, while thirty per cent. of cardiac disease developed after other methods were initiated for the relief of the constitutional disease. The while question is one bristling with difficulties, and still requires investigation. But it is reasonable to infer that as the salicylates promptly arrest the articular inflammation and allay the fever of uncomplicated acute rheumatism, they will prevent the visceral inflammations so apt to develop when the disease runs its course uninfluenced by treatment; but experience has shown that they do not control or arrest inflammation of the heart or pleura or the attendant pyrexia, although capable of subduing the articular inflammation and the pyrexia that accompany it. The best authorities are divided on the subject. Maclagan (loc. cit.), while admitting that the salicyl compounds do not ward off cardiac complications, or cure them when they exist, maintains that their existence is an additional reason for giving these remedies freely and in large doses. Broadbent (Lancet, 1882, i, 138), while believing in their protective influence "when brought to bear upon the fever in the first days of its existence" finds in the presence of any cardiac inflammation a reason for at once discontinuing those remedies. Flint (Med. Rec., 1882, 66) believes that rheumatic endocarditis and pericarditis have become more common since the

introduction of the salicylate treatment than when the alkaline method was relied upon almost entirely, and advises (Pract. Med., Ed. 5, 1098) the administration of alkalies with the salicylates to protect the heart. Vulpian (On the Mode of Action of the Salicylate of Soda in the Treatment of Acute Articular Rheumatism, Paris, 1881, 11) thinks the protective power in question probable, but not established; while Homolle (New Dict. of Med. & Surg., xxxi, 1882, 648) is of the opinion that cardiac affections are really less frequent in patients treated with salicylate of soda than in others. The development of hyperpyrexia is not always prevented by the salicyl remedies, even when they have produced their full physiological effects. Fagge (loc. cit.) endeavours to explain away the two cases of hyperpyrexia which occurred under Greenhow and the other two which happened amongst the cases tabulated by himself, and remarks that if the temperature should fall under the use of salicylic acid, and then should change its course and rapidly attain a dangerous height, that would really show that the drug is sometimes incapable of preventing the occurrence of hyperpyrexia. This actually happened in one of Powell's two cases (Lancet, 1882, i, 135), and the patient died suddenly at a temperature of 107. F. In Greenhow's (Trans. Clin. Soc., xiii, 264) first case the patient had been taking the salicylates for four days, and was deaf and delirious when the temperature became 105.8. F. Finney (Brit. Med. Jour., ii, 1881, 932) reports a case in which ninety grains of salicine were given daily for two days, and two drachms on the third day, when pericarditis set in, and on the fourth day hyperpyrexia supervened. Haviland Hall (Lancet, 1881, ii, 1082) records an instance in which the temperature fell from 103.5. to 100.6. F. after twenty-grain doses of salicylate of soda, every three hours, taken for two days; on the third day the drug was given every four hours; the temperature rose in the evening to 103.4., and on the next day it rose rapidly to 108.7. F., and the patient became delirious, but recovered rapidly after two baths. Similar instances have been reported (Med. Times & Gaz., 1876, ii, 383). Pericarditis is not always present when hyperpyrexia arises during the administration of salicylic acid; it was absent in Powell's cases, is not mentioned in Hall's, and did not ensue in one of Greenhow's, until two days after the temperature had reached 105.4. F. However, either pericarditis or pneumonia is very frequently present when the temperature is excessive. It is generally admitted that the salicylates do not control hyperpyrexia once it exists. Notwithstanding the prompt removal of the pain and reduction of the fever by the salicyl compounds, the average duration of acute articular rheumatism is not very considerably lessened by those remedies. Thus, of Hood's (Lancet, 1881, ii, 1119) three hundred and fifty cases treated by salicylates the average duration of the illness was 35.95 days as against 38.75 under other methods. The average time spent in bed by Warner's three hundred and forty-two cases was 19.5 days under the salicylates, and by three hundred and fifty-two patients under other remedies 23.5 days. Both estimates show a curtailment of the disease by the salicylates of three to four days only, which is not a very material improvement on former methods. It would also seem that the salicylates do not materially alter the time spent in hospital by

rheumatic patients;some evidence indicates that they actually prolong that period.The following are the average residences in hospital under the salicylates,according to several authors,and they are remarkably uniform with two exceptions:Coupland,thirty-six days;Warner, 34.9;Hall,thirty-four;Southey,32.5;Broadbent,31.2;Powell, thirty-one;Finlay and Lucas(Lancet,1879,ii,420),29.7; Pwen,twenty-three;Brown(Bost.Med.& Surg.Jour.,Feb.,1877), 21.9;or a general average of 30.4 days for the salicyl remedies.Under full alkaline treatment:Owen,twenty-six days;Dickenson(Lancet,1869,i),twenty-five;Fuller(Practitioner,1869,i,p.137),22.2;Blakes(Bost.City Hosp.Reps., S.1),twenty-four;or a general average of 24.3 days for full alkaline treatment.And if to these we add Finlay and Lucas's results,27.7 days,under but two to three drachms of alkaline salts in the twenty-four hours - a quantity only the fourth of that given under the full alkaline method - the general average residence in hospital under alkaline treatment was but 25.4 days - that is,five less than under the salicyl compounds.These several estimates of the time spent in hospital under the salicyltes,with the exception of Owen's and Brown's, correspond closely with that of the time spent by Gull's and Sutton's patients under mint-water(32.8 days),although the general average of them falls short of the latter by 2.4 days.Hood's third table(Lancet,1881,ii,1120)shows that under the salicylate method 25.7 per cent. remained in hospital beyond forty days,and thirty-nine per cent. under other methods,and that about fifty per cent.more were discharged within twenty days under the other methods than under the salicylate.These statistics favour Greenhow's opinion that patients treated with salicylate of sodium regain their strength slowly,and are long in becoming able to resume their ordinary occupations.Some allowance,however,must be made for the precautions against relapse under salicylates observed in hospitals since the great tendency thereto has been recognised.Certain unpleasant or toxic effects are produced by salicylic acid and salicylate of sodium;such are nausea,vomiting,abdominal pain,frontal headache, tinnitus,incomplete deafness,vertigo,tremor,quickened respiration,very rarely amblyopia and even temporary amaurosis,and not infrequently delirium.A feeling of prostration and general misery is not uncommon.These phenomena of salicylism are in great measure to the dose employed,but they have followed moderate doses, owing sometimes to idiosyncrasy,and perhaps frequently to retarded elimination consequent upon previous disease of the kidneys or disturbance of their finction by the salicylic acid or its salt.Those agents are usually completely eliminated in forty-eight hours,but in one of Powell's(Lancet,1882,i,135)cases excretion was not completed before the fifth day,and not before the eighth in Byanow's case(Centralbl.f.Chir.,1877,809). Possibly uraemia may in some cases cause the delirium, as suggested by the observations of Da Costa(Amer.Med. Jour.,Vol.lxix) and Ackland(Brit.Med.Jour.,1881,i,337). The delirium,which may be violent or not,is often preceded by dryness of the tongue,restlessness,and rapid breathing.Impurities in the acid may account for the inconstancy with which delirium has been noticed by different observers.While but two instances in eighty-

two cases were met with by Coupland,three out of ninety cases by Broadbent,and three out of one hundred and nine by Brown(loc.cit.),Charles Barrows(Med.Rec.,April 29, 1882,456)encountered eight instances in twenty-eight cases.In one of these a boy of eleven became delirious in eighteen hours,having taken ten grains of salicylate of soda every three hours.In another instance the drug had been given in full doses for five days before the deliriøum manifested itself.These phenomena of salicylism rapidly disappear when the drug is stopped,and delirium has not always recurred on its resumption.They are less frequent in children,in whom elimination by the kidneys takes place bery rapidly and a marked tolerance of salicyl compounds exists.Occasionally more serious effects appear to be produced by the salicylates,owing to their direct action on the heart,impairing its power,as evidenced by the feeble impulse and sounds,increased frequency of the pulse,and diminution of the arterial pressure.But,notwithstanding the very large number of cases of acute rheumatism that have been treated by the salicyl compounds,very few clear instances of their toxic action on the heart have been recorded,and even in some of these there were other conditions present that may have played some part,perhaps a chief part,in the production of cardiac failure. In Greenhow's(Trans.Clin.Soc.,xiii,p.266,c.iii)case the autopsy revealed a dulated fatty heart and slightly granular kidneys,and the cardiac failure coincided with a fall of the temperature to 97.F.Goodhardt's(ibid.,p. 123)patient died in nine hours after beginning the salicylic acid,of which she took but one drachm,in divided doses,every three hours.The pulse rose rapidly to 160; she was restless and moaning,but died quietly and suddenly.Recent pericarditis,with one or two points of fatty degeneration of the heart's substance,and sound kidneys were found.The reporter of the case inclines to the opinion that the acid produced sudden collapse and cardiac failure,while Bristowe referred them to the rheumatic poison itself.Hoppe Seyler(Quoted by S. Seille,- On Salicylate Medication in Rheumatism,Paris,1879,p.54) says that he once gave five grammes of salicylic acid to a child of seven and a half years affected with articular rheumatism;shortly afterwards there occurred deafness,agitation,profuse sweating,dyspnoea,and finally fatal collapse.The condition of the heart and kidneys before and after death he does not narrate.Weber(Trans. Clin.Soc.,1877,x,p.70)describes an instance in which fifteen grains of salicine,given to a woman of twenty-seven,produced in thirty-four hours a rapid fall of temperature from 103. to 96.F.,accompanied by delirium and serious,but not fatal,collapse.It is well to remember that a similar failure of cardiac power is occasionally observed in other fevers when rapid defervescence occurs, although the salicyl compounds have not been taken;and it is certainly necessary to give these remedies cautiously,and often to administer alcohol with them,when the heart's action is at all enfeebled by protracted pyrexia and pain,or by inflammatory or degenerative disease of its substance or envelope.Indeed,if severe cardiac inflammation obtain in rheumatism,the remedies are powerless and perhaps unsafe.The sudden reduction of the temperature when much exhaustion obtains, even inthe hyperpyrexia of rheumatic and other fevers,

whether by salicylic acid or quinine or the cold bath, may be attended by fatal collapse and death from heart failure. Instead of the frequent weak pulse above mentioned, one not infrequently finds that salicylate of sodium renders the pulse very slow, laboured, and compressible, and generally at the same time the temperature has been considerably reduced below what it has been. Salicine is much preferred by Maclagan (loc.cit.) to salicylic acid and to salicylate of soda, on the grounds that it is a bitter tonic and produces less debility and more rapid convalescence than those agents, and that it never produces delirium nor depresses the heart's action. Ringer (Handbook of Therapeutics) and Charteris (Brit.Med.Jour., 1881, i, 229) state that they have never seen salicine, even in large doses, cause delirium; and Gairdner (Lancet, 1882, i) has not found it produce any unfavourable symptoms. On the other hand, Greenhow (Trans. Path.Soc., xiii, 262) found that marked depression of the cardiac power ensued in four out of ten cases whilst the patients were taking salicine, and entirely subsided after it was discontinued. Further careful and extended observation is needed before the relative value of salicine and salicylate of soda can be reliably stated. It is probable that the salt is more active and prompt than the bitter principle; and this, with the greater cheapness of the former, may perhaps account for the more general employment in hospitals of the salicylate than of salicine. Writers are not agreed regarding the doses of these agents in rheumatic fever; Maclagan, Stricker, Fagge, Broadbent, Ringer, Flint, Sée, recommend large doses at short intervals at the outset, with the view of getting the patient rapidly inder the influence of the drug. Maclagan gives salicine ʒi - ii at first hourly, then every two hours as the acute symptoms begin to decline; after the second day he allows twenty to thirty grains every four hours for two or three days; "and for a week of ten days more that quantity should be taken three times a day. Stricker, Fagge, Broadbent, and Sée recommend about twenty to thirty geains of salicylate of sodium every hour or two for six doses (two to three drachms in the day), and Ringer would employ ten grains hourly, and if in twenty-four hours this dose has not either modified the disease or produced its characteristic symptoms, he would increase it to fifteen and then to twenty grains hourly. Owen's (Lancet, 1881, ii) results, on the other hand, show practically no difference in the duration of pain and pyrexia and in the average duration of illness from the commencement, whether three drachms or two drachms or one and a half drachms were given every twenty-four hours; and Young (Dubl.Jour.Med. Sci., 1880, 193) found that ten to fifteen grains every one, two, or three hours are sufficient. Indeed, I have obtained exceptionally good and exceptionally indifferent results under similar doses. The method that I use in practice is to give about fifteen grains every two or three hours, according to the severity of the case and until the articular pain and pyrexia are relieved. After the pain and pyrexia have yielded, the remedy should be continued in smaller doses, say ten to fifteen grains, three or four times a day, according to the severity of the case, for eight to ten days longer, to prevent relapse, and during this period exposure, exercise, and dietetic excesses must be carefully guarded against.

The salicine itself may be given dissolved in milk or enclosed in wafers; the salicylate of soda, in a solution of any aromatic water, to which extract of liquorice or syrup of lemon and a few drops of spirits of chloroform may be added. It is sometimes useful to add a little rum to the mixture, as is done abroad. Should severe cardiac inflammation exist, and, even though not severe, should there exist signs of failure of cardiac power, salicylates and salicine had better be avoided. If the secretion of urine diminish considerably under their use, or haematuria supervene, or organic disease of the kidneys exist, they should be used cautiously, and may require prompt suspension. Stimulants, and especially alcoholic ones, should be combined with them in the event of the development of marked debility being observed.

Salophen, which is a tasteless and crystalline substance composed of salicylic acid (51 per cent.) and acetylparamidophenol, may be given in daily doses of a drachm (fifteen grains every three or four hours); and it may be substituted for the salicylate of sodium if the latter produces gastric disturbances after a few days' treatment; in the opinion of some, it is almost specific in its effects, though my own experience with it have not established the claims originally made on its behalf by enthusiastic advicates.

Aspirin is another of the substitutes for salicylate of sodium; it is the product of the action of acetic anhydride on salicylic acid, and is described as passing through the stomach unchanged and to liberate free salicylic acid in the intestine. The best dose is fifteen grains in syrup and water, and a drachm and a quarter of it may be administered in the twenty-four hours. It certainly does not taste so nasty as salicylate of sodium, and I have found that it acts quite as well and in certain instances better than it. I have never seen it produce gastric or intestinal disturbances or collapse-symptoms, it never produces renal irritation, and none of the toxic phenomena of the drug for which it is a substitute have been observed.

WINTERGREEN

Very satisfactory result not infrequently are obtained by the use of the oil of wintergreen, which is a substitute for salicylate of soda. It is itself a methyl salicylate (ninety per cent.) plus terebene (ten per cent.). Its officinal name is oleum gaultherium, and it is given in doses of ten to fifteen minims every two hours except during sleep, and in severe cases of rheumatic fever during the twenty-four hours, either by floating the oil upon a wineglass of water or milk or in capsules or upon lumps of white sugar. It resembles in its influence upon acute rheumatism very closely the sodium salicylate, for which it may be substituted in certain cases; and there are some who have affirmed that it is quite as effectual, pleasanter to take, and free from the intoxicating properties of the salt and the salicylic acid. Like the salicylate, it should be given during the convalescence from the disease.

SODIUM BENZOATE.

This has been recommended, in as large doses as two or three drachms a day, as a substitute for sodium salicylate. I have never tried it, but observe that others claim for it that it does not produce any

gastric disturbance,is free from the toxicity of the salicylates,though quite as remedial as they.

OPIUM.

Prior to the introduction of the salicylate treatment opium was largely used for the relief of the pain of this disease;it also benefited the restlessness and nervous excitement of severe cases,and cardiac complications were not infrequently also relieved.For these indications it may therefore be given in one or other of its numerous forms,care being taken to counteract the constipation induced by the usual methods

ALKALIES.

The alkaline treatment enjoyed a widespread favour before the overwhelming advantages of the salicylates became generally known;and there are still some who maintain the excellence of the alkalies,if not their superiority,over the salicylates,in protecting the heart against the recurrence of rheumatic troubles. The alkaline method has,unfortunately,been made to include two distinct methods of administering the salts composed of potash and soda and the vegetable acids, carbonic,tartaris,etc.,namely,that in which about half a drachm of one of these salts is given three or four times a day;and the other known as Fuller's method,in which large doses are prescribed,so that froman ounce to an ounce and a half is given in the fort twenty-four hours,with the view of rapidly rendering the urine alkaline,and if possible the perspiration also;for one may often produce the former effect in less than twelve hours,and yet find the perspiration still capable of reddening litmus paper on the second,and even the third,day and later.A disregard of the essential differences existing between these two methods of emplcying alkalies in rheumatic fever may partially account for the differences of opinion existing as to the value of the alkaline treatment,and for the difference in the statisticalresults thereof published by various observers - remark applicable to other methods and statistics also.Fuller commonly ordered every three or four hours sodium bicarbonate(one and a half drachms)and acetate of potassium(half a drachm)dissolved in three ounces of water and rendered effervescing at the moment of administration by the addition of an ounce of lemon-juice or half a drachm of citric acid.As soon as the urine presents an alkaline reaction,which is usually the case in twelve to twenty-four hours,the quantity of the alkali is reduced by one-half,or to about eight drachms,during the succeeding twenty-four hours,and provided the urine continues alkaline to three drachms on the third day. On the fourth day and subsequently only a scruple to half a drachm of alkali is given three times a day,sufficient to keep the urine alkaline,and to each dose are added three grains of quinine dissolved in lemon-juice; and this combination is continued until convalescence sets in.An aperient pill is given whenever needed,but is administered"only under conditions of extreme nervous irritation!"The method is not an exclusively alkaline one.While,as we have seen,the average duration of pyrexia and articular pain under the salicylate treatment is about five days,under moderate alkaline treatment, according to the statistics of Finlay and Lucas(Lancet, 1879,ii,420),the average duration of pyrexia was 10.3 days and of articular pain 12.2 days,and of Owen(ibid.,

1881,ii,1081)6.5 days for the first and eight days for the second,or a general average for the pain and pyrexia together of 9.25 days,or about 3.85 days longer than under the salicylate treatment.Nor can it be said even of the full alkaline plan that the first or second dose frequently relieves the articular pain like a charm.On the other hand,it has been already shown that the average time spent in hospital was five days less under the full alkaline plan than under the salicylate method of medication.

The following statistics may be cited by way of illustrating the relative power of the salicylates and of full alkaline treatment in protecting the heart.The percentage of cases in which cardiac disease set in after the salicylate treatment began was,according to Powell,18.75;according to Haviland Hall,37.1;according to Finlay and Lucas,11.60;Southey,8;Brown,4.76;Jacobi, 3.35,or a general average of fourteen per cent.;whereas cardiac disease developed after the alkaline treatment had commenced in 13.6 per cent.according to Blake(Bost. City Hosp.Med.& Surg.Reps.,1870,S.1);in 10.7 per cent. according to Dickinson(Lancet,1869,i);in seven per cent. according to Owen;in 6.6 per cent.according to Finlay and Lucas;and in two per cent.according to Fuller - making a general average of only 7.8 per cent.These statistics would seem to indicate that it is improbable that a combination of sodium salicylate,with full doses of bicarbonate of sodium or chlorate of potash,will give better results in the treatment of acute rheumatism than either of those classes of remedies singly.Indeed, various observers have advised such combinations,and Bedford Fenwick has stated,as a result of his experience in thirty cases,that if,after giving a free purge,followed by scruple doses of sodium salicylate hourly for six hours,that salt be stopped,and in twelve hours afterwards half-drachm doses of citrate of potassium be adminitered every four or six hours until the saliva becomes alkaline,relapses will be extremely rare,and that this is the best and safest and most successful method of treating rheumatism in its acute and subacute forms.

QUININE.

This may be given in divided doses to the extent of fifteen to thirty grains in the twenty-four hours;the drug is much used abroad in the early stages of the disease,during the course of and on the occurrence of relapses,in acute,and especially febrile polyarticular rheumatism.It is claimed by Briquet,Monneret (Gout and Rheumatism,Paris,1857),Legroux,and others that although a not a specific for the disease it moderates the general disturbance,diminishes the local affections, and even retards the development or lessens the gravity of the cerebral symptoms:that,although it does not control the cardiac inflammations,it is not contraindicated by them.Garrod strongly advocated full doses of quinine in this disease,but he mixed the drug,in five-grain doses,with half a drachm of bicarbonate of potassium,a little mucilage,and spirits of chloroform,and gave it every four hours until the fever and articular affection had completely abated.Sufficient facts have not been published to permit of the formation of a reliable judgment as to the actual or the comparative value of either the simple quinine or the quinine-alkaline treatment of acute and subacute articular

rheumatism.There can be no doubt as to the value of quinine to meet certain conditions incident to the disease, such as debility,lingering convalescence,periodical relapse,excessive perspiration,failure of appetite,and perhaps,in some instances,high temperature.Barclay has found(St.George's Hosp.Reps.,Vol.vi,p.111 et seq.)quinine of much service when depression has followed the long continuance of the alkaline treatment and is attended with alkaline urine and a deposit of earthy phosphates. It may be taken by the rectum if intolerable to the stomach.

IODINE.

Potassium iodide,in five-grain doses combined with ten or fifteen grains of potassium bicarbonate,is sometimes of service in lingering subacute cases which have resisted the action of moderate doses of the salicylates;but it should be avoided in the early stage and in cases of severe type.Some of the earlier writers used it largely.Greenhow(Lancet,1882,i,913)treated forty-three cases with the iodide and quinine,and states that his experience of this method contrasts favourably with that of salicine and salicylate of soda.However,pneumonia supervened in three cases while under treatment; cardiac inflammation arose in six cases(equal to fourteen per cent.)after admission;single relapses of short duration occurred in twenty-one per cent.;and,excluding two cases in which the treatment was discontinued soon and seven very mild cases,the remaining thirty-four cases were on the average each thirty-six days in hospital.Under this method relapses were less frequent (twenty-one per cent.instead of twenty-six per cent.), and stay in hospital longer(thirty-six instead of thirty days),than under that by the salicylates;but the number of cases treated is too small to base a final opinion upon.He prescribed five grains each of iodide of potassium and carbonate of ammonia three or four times a day,and two grains of quinine with three of the extract of hyoscyamus in pill as often.This method,in principle at least,resembles that recommended by Da Costa,who administers in uncomplicated cases bromide of ammonium in fifteen to twenty grain doses every three hours,and as soon as the acute symptoms have disappeared follows it by quinine in fair doses.

ANTIPYRINE.

This drug is far too depressing for general use in rheumatic fever cases.

PHENACETINE.

The same remark applies to this remedy and most of the other much-advertised antipyretics.

TRIMETHYLAMINE.

This substance,erroneously called propylamine, was recommended as a remedy for acute and chronic rheumatism by Awenarius(Med.Zeit.Russlands,1858,No.6),of St.Petersburg,in 1856,by Gaston,of Indiana,in 1872,by Dujardin-Beaumetz(Union Méd.,1873,6 & 7;Gaz.Hebd.,1873, No.13 et seq.),in 1873,and Peltier in 1874,as well as Spencer in 1875;it has not been much employed,especially after the introduction of the salicylate method of treatment.It appears that in a considerable proportion of cases the articular pains have subsided in two or three days under its employment,and then the temperature has declined,but the visceral complications have not been prevented.From four to eight minims of the drug in an

ounce of peppermint-water,with a drachm of syrup of ginger,may be given every hour or two,the intervals to be increased as the pains diminish.When the pain has quite ceased the drug may be stopped and quinine take its place.It deserves further study in this disease,and Shapter,of the Exeter Hospital(Brit.Med.Jour.,1881,p. 1012)says that he is so convinced of the value of propylamine that salicylic acid has not fully commended itself to him.

COLCHICUM.

This drug appears to be without value in the treatment of rheumatic fever - to relieve the pains and general discomfort of which it must be given in such large doses as to induce derious depression and endanger life.

GUAIACUM.

This agent,acting by stimulating the cutaneous function,is chiefly of value in the chronic form of the disease.

MISCELLANEOUS MEASURES.

Lemon-juice,perchloride of iron,mineral acids, purgatives,bleeding,aconite,veratria,and so forth are indicated for the routine treatment of this disease.

SYMPTOMATIC TREATMENT.

The general treatment of the disease having been described,it is now necessary to consider the treatment of its visceral manifestations and some of its more important incidental symptoms and complications.In all cases of acute articular rheumatism it is our primary duty to employ those measures as early and as deftly as possible which in the present state of knowledge appear to promptly relieve the pyrexia and articular symptoms, and lessen the tendency to,but do not altogether prevent,the visceral complications.Such measures have been already said to be the administration of the salicylates and alkaline salts together in full doses,and the observance of certain dietetic and hygienic details.

CARDIAC AFFECTIONS.

PERICARDITIS.

The most important part of the treatment of inflammation of the pericardium is absolute quiet,both of body and mind,and especially is this true after the stage of effusion has been reached.The least exertion or emotional excitement may induce fatal syncope.Under no circumstances should the patient get out of bed,and all persons who are necessary to the welfare of the sufferer should be excluded from the sick-room.In the early stage of the lesion an attempt should be made to control the inflammatory process.Opinion is divided as to whether hot or cold applications are the more efficactious.Not a few observers advocate the use of cold, applied in the form of an ice-bag,changed as often as may be necessary,or Leiter's coil.They contend that the intensity of the inflammation is lessened,that the heart's action is steadied,and that the pain is relieved.Many patients,however,cannot endure the application of cold,and with them hot anodyne poultices must be substituted.The diet should be light and nourishing, consisting largely of milk and eggs.Beef,mutton,and chicken-broth may be given at intervals if desired, but it must be remembered that they are stimulants rather than foods.In sthenic individuals the frequency and force of the heart's action may be controlled by

veratrum viride or aconite,but their action must be closely watched,and must never be carried to actual depression.The treatment of pericarditis must be supporting rather than depressing or depleting.For this reason general bloodletting is admissible only in sthenic cases.The application ot ten or twelve leeches to the praecordium is often followed by marked relief in these cases;the pain diminishes and the heart becomes more quiet.I am of the opinion that opium is to be relied upon more than any drug in the first stage of the disease,and would advise the use of small doses, repeated as often as necessary to relieve the pain, steady the heart's action,and quiet the patient.Restlessness is especially likely to manifest itself or to increase at night,and opium affords the patient quiet and sleep.It may be administered in the form of powdered opium,compound ipecachanha powder,or as morphine hypodermically.It is never advisable to bring the patient into a state of semi-narcotism.Chloral has been advocated as a substitute for opium,but is objectionable on the ground that it is a cardiac depressant.Hyperpyrexia should be treated by sponging the body and limbs with tepid water or cold water,or,if necessary,by the cold pack.Interhal antipyretics must be avoided,because of the cardiac depression which they induce.If treatment with the salicylates,oil of wintergreen,etc.,has been induced,they must be discontinued as soon as the pericarditis is discovered.They are not only useless as remedial agents,but may be productive of actual harm from their depressing action on the heart.The alkaline treatment should be substituted.Free stimulation is called for early when there is great restlessness and cerebral excitement,unless the alcohol is badly borne. Whisky is to be preferred,but brandy or wine may be given in sufficient quantities to support the patient. In any case alcohol may be prescribed as freely as the requirements indicate.When the heart's action is turbulent,digitalis,in the form of the tincture or infusion, is of service in reducing the frequency and increasing the force of the contractions.As the liquid effusion accumulates and the heart becomes embarrassed,stimulation with alcohol or digitalis should be pushed until its action is under control.If this should fail and there is imminent danger of paralysis of the heart from compression,paracentesis of the pericardium should be resorted to without delay.The relief afforded by this operation is often marked.The question of the performance of paracentesis in any case concerns not so much the actual quantity of liquid effused as the rapidity with which the effusion takes place,since a relatively small quantity rapidly effused may cause such cardiac,and secondarily such respiratory,embarrassment as to demand immediate surgical interference.It is said that,when pericarditis is complicated with pleurisy with effusion, the dyspnoea,which in reality is cardiac,may be relieved at times by tapping the pleural cavity.The point usually selected for the insertion of the aspirating needle is the fifth left interspacemabout two inches from the median line of the sternum.Special instruments have been devised,with the idea of preventing wounding of the heart,but an ordinary aspirating needle,provided it be sufficiently large,will answer the purpose in the majority of cases.Since the heart,as a rule,is forced

back from the chest wall by the accumulated liquid, and since it tends to sink farther with the patient in the recumbent posture, there is but little danger of wounding it until the greater part of the liquid has been drawn off. Should the needle penetrate the ventricular wall in any case, the accident will be made known immediately by violent moving of the needle. Such an accident is not necessarily attended with any untoward effect. The ventricles have been entered and blood withdrawn without the production of harm. It must be mentioned, however, that death has occurred from tearing the thin wall of the ventricle with the point of the needle. Hence it is advisable to enter the pericardial cavity only so far as may be necessary to draw off the liquid. This may be determined readily by drawing on the piston of aspirator as soon as the point of the needle is well under the skin, and preserving the vacuum thus formed until the liquid is reached. Surgical cleanliness is necessary in performing the operation for fear of inoculating the pericardial cavity with pyogenic organisms, and converting a sero-fibrinous into a purulent effusion. Before introducing the needle the chest wall should be thoroughly disinfected. The skin must first be washed with soap and warm water to remove any fatty matters that may adhere to it, then with alcohol, and lastly with ether. After the liquid has been withdrawn and before the needle has been removed a piece of rubber plaster should be fixed by one side to the chest wall in position to cover the site of puncture as soon as the needle is removed. In case the effusion reaccumulates, repetition of the operation may become necessary. The absorption of any effusion which has not demanded paracentesis may be hastened by the use of diuretics, such as potassium acetate or potassium citrate combined with the infusion of digitalis, and hydragogue cathartics, as sodium phosphate, sodium and potassium tartrate, etc. But these drugs, especially the potassium salts, must be used with care, for potassium and sodium are cardiac depressants. The patient is in a weakened condition and the heart is already depressed by the disease. Calomel in small doses is to be recommended in certain cases. Occasionally when absorption is long delayed removal of a portion of the effusion by aspiration will hasten the disappearance of the remaining liquid by natural means. In these cases it is supposed that the tension on the pericardial sac closes the lymphatic channels, as in pleurisy with effusion, and that when this is taken off absorption proceeds normally. Potassium iodide in doses of forty grain a day is said to be of service in the promotion of delayed absorption. For the same purpose blisters to the praecordium are to be recommended. Quinine in tonic doses, four to six grains a day, and iron are indicated in this stage. As soon as the patient's condition will allow, a more generous diet should be arranged, including the more easily-digested meats. Purulent pericarditis demands the free opening of the pericardium, washing out, and draining.

ENDOCARDITIS.

The prophylactic treatment of simple acute endocarditis consists in counteracting rheumatic tendencies by a restricted diet, moderate exercise in the open air, and close attention to the rules of general

hygiene.Cod-liver oil should be administered to young subjects of this class,and flannel should be constantly worn next the skin in order that the surface of the body may be protected from sudden variations of temperature.On the occurrence of the slightest rheumatic symptoms the individual should be kept in bed and his diet restricted to milk containing a small quantity of bicarbonate of soda.It is doubtful if the salicylates exert any prophylactic influence against the disease.My own opinion if unfavourable to their exhibition to this end. When endocarditis is once established,no drugs seem to have any controlling influence over it.The temperature of the sick-room should be kept between 70.and 75.F.,the patient restricted to a milk diet,and the urine kept as near neutral as possible.If the heart is irregular in its action and the patient restless,from a twentieth to an eighth of a grain of the acetate of morphia should be administered at regular intervals,sufficient to keep the patient in a quiet,comfortable state.All applications over the praecordial space,except such as will maintain an equable temperature of the surface,have seemed to me to be not only useless,but harmful,especially cold applications.It is to be remembered that this inflammation rarely subsides within four weeks,and this course therefore should be rigidly enforced during that period.Too great care cannot be exercised during convalescence to protect patients against exposure to changes of temperature and excessive fatigue.The patient should be careful what he eats,and after meals he may be advantage have small doses of iron and cod-liver oil.

In malignant endocarditis there seems to be no form of treatment capable of exerting a special influence over the course of the disease.Cold applications are not possessed of signal utilitarian properties,and they make the patient feel even more uncomfortable than before.But stimulants should be freely used,and opium in sufficient quantities to relieve the depression which is so marked a feature in its early period.These cases, however,as stated before,are practically always fatal.

CHRONIC VALVULAR LESIONS.

The treatment of chronic valvular disease must be varied to suit the stage of the affection and the exact conditions present.It must be remembered that no medication or other treatment at out disposal will remove the valvular lesion,and that all of our therapeutic endeavours should be directed to the establishment and maintenance of compensatory power on the part of the heart.If compensation be perfect,the patient requires rigid hygienic care,but no medicinal treatment,and the too frequent practice of prescribing digitalis or other cardiac stimulants,merely because a murmur is discovered, must therefore be condemned.Above all,it is the patient who must be studied,and not the murmurs.In the series of valvular events there are,first,a period of perfect compensation;second,a period when the compensation begins to fail;and third,a period of complete failure of compensation.During the first period,as stated,no medical treatment is required.The individual must lead a quiet life,free from excitement and worry.His diet must be non-stimulating and restricted in quantity to the requirements of perfect mutrition.In eating and drinking he should obey the physiological laws of health.

alcohol and tobacco should be sparinglyused,if at all. Physical exercise is essential,but it should never be excessive or prolonged to fatigue.There are no fixed rules as to bathing.Each individual must be governed by its effects.In most cases sponging the surface of the body daily with cold or tepid water is of service,for it has a tendency to keep the skin active and stimulate the surface circulation.Sexual indulgence should never be excessive,and in some instances be entirely abstained from.The patient must be preserved from exertion,mental worry,and those thingswhich disturb his nutrition. If there is a strong rheumatic tendency,the patient should take up his residence in a moderately warm,dry climate, one that is not liable to sudden changes of temperature. The first indication that a patient with chronic valvular disease is entering upon a period of commencing failure of compensation requires the most careful consideration.Undoubtedly the earliest and most positive sign of such failure is sudden attacks of dyspnoea after active physical exertion or on awakening from sleep at night.Now the patient should be made to understand his exact condition and the dangers which attend it.He should be told of the serious effects of sudden physical or strainor emotional excitement in producing cardiac dilatation,that simple overindulgence in eating or drinking or sexual excesses may lead to serious cardiac insufficiency which will never be fully recovered from.His emotions,passions,and indulgences must be kept under perfect control.Carefully regulated physical exercise is important,but it should never be carried to fatigue,and should always be followed by a period of perfect rest.All these indications can best be met in a quiet life in the country.Besides the regulation of the diet,it must be remembered that the stomach must have all the rest compatible with perfect nutrition.It is often difficult to combine both indications.Out aim must be to obtain the most perfect nutrition with the least tax on the digestive organs.Therefore,this class of patients should indulge sparingly in sugars,starchy vegetables,and animal fats.Their food should be largely nitrogenous and taken in small quantities at a time,so as not to embarrass the heart's action.If relief from the dyspnoeic attacks and the other signs of commencing cardiac insufficiency does not follow careful regulation of the diet and exercise and freedom from care and anxiety and excitement,a mild mercurial course,combined with small doses of digitalis and some saline,should be resorted to,the patient remaining in bed during its administration.Two grains of gray powder with half a grain of the extract of digitalis and twenty grains of magnesium sulphate,every four hours,will be found perhaps to be the best combination for overcoming the commencing failure of compensation. The use of digitalis during this period requires the greatest care.Harm is often done by giving more than is required to regulate the cardiac action.When inchronic valvular disease failure of compensation,indicated by dilatation of the heart's cavities and feebleness of the cardiac walls,is established,the period for active interference is reached.There is no class of cases which require sounder judgment and more experience in their management than these.While each case must be

studied by itself, there are certain general rules which may serve as a guide in their management. The same general plan of treatment suitable before the failure of compensation should be continued as far as possible.

In aortic stenosis, as soon as failure of compensation is established, not only must absolute rest be enjoined, but the patient must assume as much as possible a horizontal position. The rules in regard to a restricted diet must be rigidly enforced and all active exercise prohibited. The surface of the body should be covered with flannel, and exposure to sudden changes of temperature avoided. Frequent massage will give relief to the sluggish cutaneous circulation. The medicament which most certainly increases the force of the cardiac systole and relieves the tendency to vertigo and syncope in this condition is strychnine, in doses of a sixtieth of a grain administered before food in the morning and in the middle of the day. It will often stay the compensation for a long period. Under its influence the pulse loses its irregularity in force and rhythm and the signs of cardiac insufficiency disappear. The failing compensation of aortic incompetency is usually associated with very great cardiac enlargement and of extensive arterial changes; consequently, for a long time there will be periods of temporary loss of compensation, which will be quickly re-established by regulation of diet, mercurial catharsis, and rest; but when the period of complete failure is reached the patient has entered upon the most serious condition of valvular disease with which we have to deal, except tricuspid insufficiency. Rest in bed does not now give the same relief as in aortic stenosis; nevertheless, it is important to maintain it as far as possible, and all the other rules which have been given regarding diet, exercise, etc.; but strychnine will not so certainly or so safely increase the force of the cardiac strokes as it does in stenosis. It may be often observed that this class of patients are made worse by the use of strychnine, while digitalis not only increases the force of the cardiac systole, but slows the action of the heart and causes the pulse to become firmer, fuller, and more regular in rhythm. If the only object is to increase the power of the ventricular systole and increase the contraction of the peripheral vessels, the tincture is its most reliable form of administration. It may be given in doses ranging from five to thirty minims in the twenty-four hours, the amount in each case being determined by a careful trial, the patient remaining in bed during the time. When it acts beneficially, the dyspnoea will be relieved in twenty-four hours and the urine increased in quantity. Digitalis becomes, as it were, a food to some of these patients. If in any case of aortic insufficiency a condition of high vascular tension is reached, nitroglycerine will often give the most marked relief. One minim of the officinal solution may be given every three or four hours.

In cases of mitral disease failure of compensation will be marked by pulmonary complications, by a rapid, feeble, irregular pulse, by scanty urine, constant dyspnoea, and dropsy. The evidences of anaemia become marked in dilatation of the right heart. An important factor in the failing compensation is greater or less obstruction to the venous circulation. Everything that taxes or interferes with respiration must be carefully avoided,

and the greatest care must be maintained in diet and exercise.All the predisposing causes of bronchitis and acute pulmonary affections must be guarded against.Straining at stool must be avoided and the use of alcohol and tobacco prohibited.Gentle daily evacuation of the bowels must be maintained.The patient should avoid prolonged use of the voice,especially in speaking and singing.The most strikingly beneficial effect of digitalis in the treatment of valvular lesions is seen in cases of mitral disease.Out aim here must be to slow the pulse, make it more regular,and of higher tension.The power of digitalis to strengthen the contraction of the right ventricle when it has become dilated is even greater than its action on a dilated left ventricle.By its action on the right heart it overcomes the obstruction in the pulmonary circulation,and this tends to restore the failing compensation of the left heart.It often does this in a surprising manner,causing the most extreme dyspnoea to disappear by relieving the over-distended pulmonary vessels and for a time re-establishing complete compensation.In the administration of digitalis in mitral disease we should endeavour to obtain the desired therapeutic effect with the smallest possible dose.Five or ten drops of the tincture twice a day are often amply sufficient to accomplish all that is desired.When it is important to produce a rapid and powerful effect in cases where there is considerable dropsy,marked embarrassment of respiration,cyanosis,scanty urine,a small, irregular pulse,and a tumultuous action of the heart,much larger doses may be given.Twenty minims of the tincture or half an ounce of the effusion,every two hours for twenty-four or forty-eight hours,may be required to accomplish the desired result.Whenever in mitral incompetency complete asystolism is present and suppression of the urine is threatened,digitalis should be freely given,whether the other indications for its use are present or not.In some mitral stenoses,however,even when the pulmonary symptoms are urgent and cyanosis is extreme,digitalis should be used with great care,and its administration should be preceded by the free application of wet cups to the chest and leeches over the liver.Whenever large doses of digitalis are being administered in any valvular disease it is important that the patient should remain in bed and the bowels be kept freely open with mercurial cathartics.Sometimes,when large doses of digitalis are prescribed in mitral disease for a considerable time,the toxic effects of the drug are produced.The pulse becomes irregular and small,there may be two beats of the heart to one of the pulse,the urine will be reduced in quantity,and there may be nausea and vomiting.The urine is the best guide to the continuance of its administration:as long as it is increasing in quantity or freely secreted there is little danger of its toxic effect.If toxic symptoms occur,there is no danger attending it,for they soon vanish after the exhibition of the medicament is suspended.

Digitalis should not be administered in Tricuspid incompetecny secondary to mitral disease,where there are cyanosis and symptoms which indicate engorgement of the right heart,until the venous engorgement is relieved by venesection,by the application of leeches,or by free purgation;for digitalis and general stimulation

under such circumstances are dangerous, and may destroy a life that might have been saved. It must never be forgotten that the free use of digitalis is more harmful than anything else in cases of commencing failure of compensation in tricuspid regurgitation.

Other valvular lesions must be treated on general principles and according to the indication of the individual case.

Finally, there are certain things to be borne in mind as regard the occurrence of dyspnoea and dropsy. The former is perhaps one of the most distressing symptoms of established failure of compensation. In those cases in which digitalis fails to re-establish the compensation and the attacks of dyspnoea are frequent and extreme or continuous, morphine administered hypodermically is invaluable and should be given without hesitation. It generally gives immediate relief; the amount to be administered in any given case must be determined after a careful tentative employment. Large doses may be necessary to obtain relief in advanced cases. In those with extensive arterial changes, or where there is a pulse of high tension, a one-per-cent. solution of nitroglycerine may give relief when administered in increasing doses with digitalis. If the dyspnoea is increased by the presence of fluid in the thoracic or abdominal cavity, it should be promptly relieved by aspiration. In all cases rest in bed should be provided, which will support the arms and head and at the same time give rest to the feet and legs. Nitrite of amyl seldom gives even temporary relief to this class of patients. Regarding the second of the above-mentioned special conditions, dropsy, it may be noted that oedema of the feet is usually the first indication of commencing failure in valvular diseases. At the outset it usually rapidly disappears by the free administration of digitalis and rest in bed. If this fails, calomel and squills, combined with digitalis, in pill-form, will cause it to disappear very soon. Hydragogue cathartics are of doubtful efficacy in the treatment of these cases, and the so-called diuretic plan of treatment has not been at all satisfactory in my hands. It is never advisable to attenpt to renove the dropsy by pilocarpine or hot-air baths, lest fatal syncope should result. In the worst cases, when the patient's condition is rendered distressing by a waterlogging, general anasarca, sacrification of the skin will allow the serum to drain out and give the greatest relief. If the operation be performed antoseptically after thorough cleansing of the parts, it will not be followed by any unpleasant results. After the scarification the parts should be covered with carbolised cottonwool. The patient's position should be changed frequently to prevent distention of the dependent parts.

CARDIAC DILATATION.

We have seen that acute dilatation of the heart may arise in rheumatic fever cases; and the important indication here is to lessen the work of the heart as much as possible. This can be best accomplished by absolute rest in bed. The patient should there be kept free from mental excitement, until the cardiac symptoms disappear. When the dilatation first occurs and the heart's action is feeble and irregular, stimulants should be administered, and of these alcohol is probably the best. It should be given in doses sufficient to control

the heart.If alcohol should fail in its action,amyl nitrite,nitroglycerine,sodium nitrite,or hypodermic injections of strychnine may be tried.An ice-bag or Leiter's coil applied to the praecordia will frequently afford the patient relief from the cardiac oppression and calm the condition of the heart.In certain cases, when the venous system is overloaded with blood which the right heart is unable to carry off,venesection is of signal service.After the patient has recovered from the acute symptoms and is allowed to get out of bed,the greatest caution should be exercised against over- or sudden exertion.He should be careful not to place himself in positions which render such exertion necessary.He should not attempt to run or ascend stairs quicklyCardiac tonics and stimulants should be administered during this period with a view to assisting the action of the heart until the development of a compensating hypertrophy has been completed.The best remedial agent is digitalis in doses of five minims or more of the tincture,three times a day,as the case may require;and its action may be greatly enhanced by the administration with it of a fifth of a grain of strychnine after food.The ordinary tonics may be also prescribed.

MYOCARDITIS.

Absolute rest is an important part of the treatment of acute myocarditis.The patient must not rise from the reclining position for any purpose.Quinine and strychnine are exceedingly valuable as general muscular tonics;but cardiac stimulants,especially digitalis,should be used with care,and never in large doses.Clknical experience has demonstrated the value of alcohol in the myocarditic of rheumatic fever,and this remedy should therefore he given as soon as the first sound grows weak.In cases of cardiac abscess large doses of digitalis might favour a fatal rupture of the heart.When uneasiness is experienced in the praecordial region,cold applications will frequently bring prompt relief.For the same purpose Leiter's coils,ice-bags, or cloths saturated with iced water may be applied. In very case of rheumatic fever in which cardiac weakness is observed care must be taken to avoid undue excitement or exertion for a considerable period after the subsidence of the disease.

NERVOUS AFFECTIONS.

These include organic lesions and functional disorders.If it were possible,which it is not,to differentiate generally rheumatic meningitis from the merely functional form of so-called cerebral rheumatism,then its treatment would resolve itself into a vigorous use of the antirheumatic remedies,salicylates,alkalies,etc., and the active employment of leeches to the scalp, purgatives,full doses of the iodide and bromide of potassium,ergot,etc.If,together with the symptoms of that often obscure and comparatively rare complication of rheumatic fever,ulcerative endocarditis,there occurred severe headache,delirium,or paralysis,we might find great difficulty in determining the cause of the cerebral disturbance,and would naturally very our measures according as we suspected meningitis,embolism,or simple functional disturbance,and the treatment adapted to these several condition must be applied according to the usual well-recognised plans of the text-books,which need not be here considered.Regarding the functional

disturbances of the nervouscentres,which are the ordinary forms encountered in rheumatic fever,they may be divided into two categories,namely,those with hyperpyrexia,and those preceded,accompanied,or followed by that complication.In the first class of cases,when any evidence of disturbance of the nervous system,delirium, restlessness,taciturnity or talkativeness,insomnia or somnolence,deafness,tremulousness,vacancy,stupour,or what not,occurs in rheumatism with but a moderate temperature,101. to 103.F.,while we anxiously watch the temperature from hour to hour,prepared to combat any tendency to hyperthermia the moment it is doscovered,we endeavour to control the cerebral disturbance as in other febrile affections,but with greater diligence, knowing that in this disease these nervous symptoms very often precede hyperpyrexia.We persist with the salicylates to reduce the rheumatic element of the affection, employ remedies to control the cardiac or pulmonary inflammations which are so frequent,in such circumstances,sustain the general powers by food,wine,and quinine,if,as frequently happens,there are evidences of failing strength,and meet any other special indication that may arise.For example,we procure sleep and allay motor and mental excitement by opium or chloral and by evaporating lotions or the ice-cap to the head.We reduce temperature,allay restlessness,preserve the strength, and promote sleep by lightening the bedclothes,drying frequently the entire surface of the body if it is perspiring freely,or by sponging it with tepid water hourly if dry and hot.We act on the kidneys,bowels,and if necessary the skin,if from the scantiness of the urine or other evidence we suspect uraemia.Should these means fail and the delirium and other symptoms which occur in cerebral rheumatism continue,and especially should they be severe,it would,I think,be proper to employ the methods that are now resorted to when hyperpyrexia accompanies those symptoms;for patients suffering from cerebrospinal disturbance or rheumatic fever,although unattended by hyperthermia,do die if these symptoms continue.Moreover,the hyperthermia may at any moment supervene;it is itself perhaps just as much a nervous disturbance as delirium,and apt to succeed the latter.It was inthese very cases in which delirium preceded the hyperpyrexia that the committee to be presently mentioned found the highest mortality.If along with these nervous symptoms the articular pain or the sweating disappear suddenly,or if the pulse suddenly increase in frequency without demonstrable increase of cardiac mischief, there is reason to acticipate the supervention of the hyperpyrexial condition.In the second class of cases, when the cerebrospinal disturbance is followed,preceded, or accompanied by hyperpyrexia,there is one indication for treatment which dominates all other,and that is the prompt reduction of the hyperthermia.The terrible danger of this condition in rheumatic fever is known to all practitioners who have had much experience of the disease.Wilson Fox says that he never knew a case recover after a temperature of 106.F.unless under the use of cold,yet that is not an alarming temperature in intermittent or relapsing fever,and is often recovered from in typhoid fever.Largely through the efforts of Wilson Fox(Treatment of Hyperpyrexia,1871; Lancet,1871,ii), Meding(Arch.f.Heilk.,1870,xi,467),H.Thompson(Brit.Med.

Jour.,1872,ii; Lancet,1872,ii; Clinical Lectures,1880), H.Weber(Trans.Clin.Soc.,v,136),I.Andrew(St.Bart.Hosp. Reps.,x,337),Maurice Raynaud(Jour.de Thérap.,1874,No. 22),Black(Gaz.Hebd.de Méd.Sci.,1875),Fereol(Soc.Méd.des Hôp.,1877,June 8),and many others since then,it has been established that when the hyperthermia is removed by external cold the nervous disturbances also usually disappear or lessen very much.And thus we are brought to the treatment of the hyperpyrexia of rheumatic fever.On this important subject it will be most satisfactory and convincing to give some of the conclusions arrived at respecting hyperthermia in acute articular rheumatism by the committee of the London Clinical Society(Brit.Med. Jour.,i,82,807)."Cases of hyperpyrexia in acute rheumatism prevail at certain periods";/such excess corresponds in a certain degree,but not in actual proportion,to a similar excessive prevalence of acute rheumatism generally.The largest number of cases of hyperpyrexia arise in the spring and summer months,whereas rheumatism is relatively more common in the autumn and winter!"Whilst very little difference obtains between the two sexes in regard to proclivity to rheumatism,the proportion of males to females exhibiting hyperpyrexial manifestations is 1.8 to 1!""The cases of hyperpyrexia predominate in first attacks of rheumatic fever!""Hyperpyrexia is not necessarily accompanied by any visceral complications, but may itself be fatal.The complications with which it is most frequently associated are pericarditis and pneumonia!""The mortality of these cases is very considerable,hyperpyrexia being one of the chief causes of death in acute rheumatism!""Although present in a certain number of cases,and these of much value from their prodromal significance,neither the abrupt disappearance of articular affection,not the similarly abrupt disappearance of sweating,is an invaraible antecedent of the hyperpyrexial outburst!""The post-mortem examinations in a certain proportion elicited no distinct visceral lesions, and when present the lesions were not necessarily extensive!""The prompt and early application of cold to the surface is a most valuable mode of treatment of hyperpyrexia.The chances of its efficacy are greater the earlier it is had recourse to.The temperature cannot safely be allowed to rise above 105.F.Failing the most certain measure -viz.the cold bath- cold may be applied in various ways:by the application of ice,by cold affusion,ice-bags,wet sheets,and iced injections!No matter what may be thought of the utility of cold in the treatment of hyperthermia i typhoid fever,there is a tolerable concensus of opinion that it is our most reliable and promptest measure in those formidable cases of rheumatic fever attended with hyperpyrexia,both when alarming delirium and coma coexist and when they are absent. There are numerous details to be observed in the employment therapeutically of cold which need not here be entered into.Suffice it to say that,besides the cold bath (70. or 60.F.)which the committee in question regards as the most certain,the tepid bath(96. to 86.F.)is employed by Fox and regarded as the best by Andrews;it may be cooled down to 70.F.by adding ice or cold water to it. The cold wet sheet-pack is still thought much of,like the last,in old and feeble individuals.Kibbie's method deserves more attention than it has apparently received. He pours tepid water(95. to 80.F.)over the patient's

body,covered from the axilla to the thighs with a wet sheet and laid upon a cot,through the open canvas of which the water passes and is caught on a rubber cloth beneath the bed,and conveyed into a suitable receptable at the foot of the latter.Cold bathing is contraindicated not even,according to some,when polyarthritis,pericarditis,endocarditis,pneumonia,or pleurisy exist. If much weakness of the heart exists,and it is decided to have recourse to the measure,it is advisable to give some wine or brandy before employing the bath,and perhaps while in it,and the patient should not be kept in the bath until the temperature reaches the normal,for it continues to fall for some time after his removal from the bath.If the temperature fall rapidly two or three degress in five or six minutes,the patient must be removed from it as soon as the body-heat recedes to 102. or 101.F.If it fall very slowly,the bath may be continued till the temperature declines to 99.5.F.,when he should be taken out.Should marked symptoms of exhaustion or of cyanosis arise,the bathing should immediately be stopped.After it has been found necessary to employ cold in this way,the thermometer should be used every hour,and if the temperature tend to rise rapidly again,the diligent application of a succession of towels wrung out of iced water and applied to the body and limbs,or of Kibbie's method,may suffice;but should they not,and a temperature of 103. or 104.F. be rapidly attained again,the cold ot tepid bath should be at once resumed.In severe cases of this kind a liberal administration of alcohol and liquid food is generally needed, and it is well totry antipyretic doses of quinine by mouth or rectum,although they are usually very disappointing in these cases.It is admitted that cold baths have in a few rare instances caused congestion of the mucous membrane,pneumonia,pleurisy,and even fatal syncope.This is a reason for the exercise of care and constant oversight on the part of the physician,but hardly an excuse for permitting a person to die in rheumatic hyperpyrexia without affording him at least a chance of recovery by the employment of hydrotherapeutic measures in this way.During the course of the exhibition of the salicylates,the supervention of delirium and deafness will call for their suspension and the estimation of the temperature every two hours;for one is never sure in these circumstances that hyperpyrexia is not impending.The deafness from the administration of sodium salicylate can be mitigated by the addition of dulate hydrobromic acid to the solution employed.

S e r u m t h e r a p y.

The serum treatment of rheumatic fever has of late been ardently urged.But the method is not free from danger,and doubt as to the real nature of the disease is,in the opinion of not a few observers, a decided contraindication,particularly in rheumatic chorea which is notoriously uncertain in its course. The antistreptococcic sera in many quarters advised are just as uncertain perhaps and not easy to standardise.A polyvalent serum manufactured from various strains of streptococci is expected,by those who refer the origination of the disease to streptococcal infection,to prove of service:on theoretical grounds it certainly should,provided,of course,the morbific conditions are not unconnected with a mixed infection

with other organisms producing more or less similar or allied poisons.Menzer reports success with Merk's serum in cases of subacute rheumatism,but his results have not been uniformly obtained by others.

LOCAL TREATMENT.

For the local treatment of acute aarticular rheumatism no uniform method is invariably applicable,though in acutely inflamed cases topical applications of a suitable sort afford marked relief.The first indication is to secure absolute rest of the affected joint,and anodyne applications and antiphlogistic remedies constitute the second.In order to secure rest,the articulation should be wrapped in a thick layer of cottonwool covered by oilsilk,and bound to a splint with a few turns of a light gauze bandage.This simple measure alone not infrequently gives the greatest relief from the pain.It serves the threefold purpose of fixing the joint and preventing the friction of the inflamed surfaces;it protects from jarring or accidental pressure;and it maintains a uniform temperature about the joint.If the elbow or knee is involved,the parts above should be immobilised;if it be the shoulder which is affected,the arm should be bound to the side;if the wrist,it must be laid upon a padded arm-and-hand splint. Smaller joints must be similarly protected.When the joints of the lower extremity are affected,it is often desirable to lay sandbags on either side,for in this manner perfect rest is secured and the patient is less likely to injure himself by turning in his sleep.A good supply of pillows of different sizes are of great service in easing the constrained positions of the body.As a rule,in the acute stage of rheumatism moderate flexion of the larger joints is the position in which they are most comfortable if duly supported.As the inflammation is apt to pass rapidly from one joint to another,much skill is required in the nursing of the patients in order to mitigate the pain experienced. There is but one objection to the above suggestions,and that is the impossibility of making local applications to a joint while it is enveloped in cottonwool; but many of these applications do not require frequent repetition,and may be made once for all day,or,if desirable,a hole may be left in the dressing or the cottonwool covering and the joint is then simply bound above and below to the splint.As a rule,warm applications are preferred to cold for the reduction of the inflammation and the relief of the pain,and in most cases the warmth retained by the cottonwool,as above described,is all that is necessary.Occasionally linseed poultices give relief,more particularly to the less acutely inflamed articulations.Some writers recommend the application of ice continuously to the joints until all the symptoms of acute rheumatism have vanished therefrom.Strange as it may seem,when the larger joints are most acutely inflamed few applications give so much relief as one or two active cantharidal blisters applied directly over the focus of the inflammation.They should be large,-in case of the knee two to three inches square,-and sufficiently active to withdraw an ounce or two of serum, after which the blister is to be opened and dressed and the joint wrapped in cottonwool as before.The number of evaporating lotions,liniments,and embrocations recommended for acute articular rheumatism is very

large.They constitute the chief part of the sales of proprietary and patent medicines,many being worthless, some relieving one case,some another.As might be expected,most of these applications are of more benefit in subacute and chronic cases,where their favourable use may be accompanied by friction,than they are in acute inflammation.For the latter it would seem that two remedies exceed all others,namely,oil of wintergreen and guaiacol.Their application is often followed by immediate and lasting relief,although,like all remedies,they may sometimes fail.The gaultherium oil is applied by soaking a piece of lint freely with the undiluted oil, and the affected articulation is then encased in cotton-wool.The application may be renewed once or twice in the twenty-four hours.Guaiacol may be applied in a similar manner,first diluting it with an equal part of glycerine. It is even more efficacious than the gaultherium,but it possesses an odour not unlike creosote,which is more disagreeable to many,and which has a peculiar penetrating and persistent property.Efforts to overcome this objection are not uniformly successful.If the application should cause itching or slight burning,the guaiacol may be further diluted.The drug is well known to produce a remarkable fall in temperature when rubbed into the skin in doses of half a drachm or more,which may be accompanied by collapse.Dana has reported a drop of nine degrees in the temperature in a case of typhoid fever which was produced in this manner,and an even greater fall has been produced experimentally in animals; but for some reason in rheumatism one seldom or never finds any marked thermic reaction,and it is therefore justifiable to apply the remedy quite freely.Only on rare occasions does it seem to reduce the general body temperature,and then to the extent of only two or three degrees.It is now often used and the results are usually eminently satisfactory.Methyl salicylate is also recommended;from five to a hundred drops may be applied to the affected articulation.Salicylic ointment has also been used with success.Bourget found this acid in the urine twenty-four hours after its local application,proving that much of it was absorbed.He prescribed salicylic acid,lanolin,oil of turpentine,of each ten parts,with seventy parts of lard.Numerous other topical applications have been recommended and advertised,but they need not be considered here.By means of a special instrument,the electro-thermogen,Taylor(Lancet,Nov.26,1898)has successfully employed the local application of currents of hot air.The light application to the affected articulation of the Paquelin thermo-cautery has likewise been advised.

TREATMENT OF RHEUMATIC FEVER IN CHILDREN.

A child who comes from a rheumatic family seems specially prone to contract the disease;and one who has once suffered from it is very apt to have it again. In both cases PROPHYLAXIS should not be omitted,and all due precautions taken to protect those who are thus predisposed from overheating,chilling,and over-fatigue, the great sources of rheumatism.To this end the child should not be kept too tenderly,but should be kept out of hot,close rooms,should live in a cool and even temperature,should be clothed in woolen garments next the skin,while the body is hardened by tepid salt-water

baths and vigorous friction.In a case of accidental exposure to cold or wet,brisk exercise should be taken until a free glow of warmth is experienced,and damp clothes changed at the earliest possible moment. When heated or overtired by severe exertion,standing about in cool air should be acrefully avoided,and the body protected against chill by an extra covering until it cools down again.Damp air and cold soil and variable climate should be avoided.When circumstances allow,the rheumatic child should be taken to a dry,warm climate, with sandy soil,in a locality not too woody,exposed to the free action of the sun's rays,and with a free circulation of air about.

GENERAL MANAGEMENT.

In dealing with a case of rheumatic fever i childhood it is necessary to prevent fresh chilling of the surface;to keep the affected joints at rest,so as to lessen the flow of blood there and friction of parts,and thus lighten inflammation and releive pain experienced. This applies not only to the joints,but also to the heart.Numerous observations show how important a means this is of modifying the after-effects of endocarditis. It is also to modify,if possible,by specific remedies, the fever and neutralise the irritating effects of the rheumatic poison on the fibrous tissue of joints and tendons;and likewise,to prevent,if possible,the inflammation of the endocardium and the pericardium,or,if this has set in,to minimise and arrest it;finally,to rlieve pain directly by anodynes if necessary.It may be though that many of these ends cannot be attained with certainty;and,indeed,the statements made as to the effects of remedies in rheumatism are in some respects conflicting and unsatisfactory.Nevertheless,there is evidence that most,if not all,of the objects laid down can some of them be effected with certainty and others greatly aided by the use of remedies.Should the tongue be coated or the bowels constipated,a dose of one to three grains of calomel should be given at the outset: this constitutes an excellent introduction to other forms of treatment.To insure against chill and give rest the patient should be kept in bed,either between blankets or in a flannel nightdress,until some time after the temperature had become normal and all symptoms have ceased.The diet in cases of acute rheumatism does not call for the close limitation which is usually enforced. When the temperature is raised,and acute symptoms are present,it should consist entirely of beef-tea or broth with milk.In cases of great anaemia or prostration,one or other of the reliable meat-extracts,or even raw meat-pulp itself if it can be taken,may be given as blood-restorers;and self-digesting foods,or peptonised milk, are most useful.As the fever declines,light puddings, eggs,bread-and-butter,and tea may be allowed;and thus the patient pass on meat,fish,and so forth.Sugar and sweets are open to theoretical objection.

MEDICINAL TREATMENT.

The exhibition of salicine or salicylate of soda constitutes the best method of reducing the fever and arthritis of this disease.Authorities are generally agreed as to the actuality of this,although some of them differ as to their ultimate effect on the prevention of cardiac complications and the duration of the malady.

We have seen that, according to the statistics of the Collective Investigation Committee(Report, 1888, Vol. iv, p. 75), the duration of the whole attack is 19.03 days, of the fever, 8.65 days, nad of the pain, 10.18 days, their observations being based upon six hundred and fifty-five cases. In the case of children salicylate of soda, freely administered seldom or never fails to reduce the temperature and relieve the pain of rheumatic fever in the course of twenty-four to forty-eight hours, -the Report of the above-mentioned Committee states that there were twenty-two cases out of two hundred and ninety-six treated by salicylates and salicine in which they were ineffectual, but rhe doses employed were probably too small and at too long intervals; but there are several drawbacks to its use. In the first place, it sometimes causes children to feel sick and vomit to a distressing degree. It also has a depressant effect on the heart; the pulse loses strength, and the first sound of the heart becomes faint, so that in several reported cases it was almost inaudible. It also produces in certain cases deafness, buzzing in the ears, vertigo, and delirium. In extreme instances the symptoms have become alarming, such as great prostration, violent delirium, albuminuria, and collapse. It is true that these symptoms occur much less frequently in children than in adults; still, in view of the proneness to cardiac disease in the young, it is well to such a depressant drug with caution. As a matter of fact, it is only occasionally in children that the pain and fever are sufficiently important to require its administration. If the articular symptoms are severe, however, and temperature much raised, salicylate of soda will reduce them more quickly than any other remedy. It should be given for the first twenty-four or forty-eight hours, and after that salicine should be substituted for it; for the latter drug has little, if any, of the evil properties of the former, although it appears to act through its conversion into salicylic acid in the blood. through the action of ptyalin or other ferment. Possibly it acts less violently because more gradually passed into the circulation; it certainly produces its effect more slowly. In all but the most severe cases salicine is, then, preferable to saliculate of soda, and may be given for a day or two in doses of five to eight grains every three or four hours to a child of five, mixed with water and syrup or orange or other demulcent agent. The salicine should be continued in less frequent doses for some days after all symptoms have ceased, or a relapse is liable to occur. These remedies will serve the purpose of reducing the temperature and the arthritis, but, unfortunately, they seem neither to prevent the occurrence of cardiac inflammation or modify it when developed. There are abundant statistcs to prove this. One would have naturally supposed that a drug which cuts short the arthritic manifestations of the rheumatic poison would also prevent or arrest what are believed to be similar changes in the pericardium and valves of the heart; but this is not the case, and one not infrequently observed that the evolution of fibrous nodules, which may perhaps be taken as the external index of what is going on in the fibrous tissues of the heart, continues to occur in spite of the steady administration of the salicine. The only treatment which can claim to show good results in this respect is by alkalies.

The treatment of rheumatic fever by alkalies has already been discussed at some length;their influence in mitigating the articular symptoms and shortening their course appears distinctly favourable,although much less decided than that of salicine and the salicylates.Alkalies should,then,be given in combination,salts of soda in preference to those ot potash,as being less depressent.With the salicylate of soda,six to ten grains of bicarbonate of soda;the same with the salicine. The amount of alkali must be regulated by the condition of the urine;enough should be given to render it neutral or slightly alkaline.If,however,endocarditis or pericarditis come on,the salicylates or salicine should be at once stopped,and the alkali given in freer doses,-ten to fifteen grain every four hours,with half a drachm of syrup in half an ounce of water.In severe and obstinate cases of endocarditis or pericarditis,when there is high temperature,palpitation,cardiac dyspnoea,and distress, quinine should be given in addition,in doses of two to three grains every four hours for a child five years old.This may be done by giving ten-grain doses of citrate of soda,two grains of quinine,and five grains of ci-tric acid;or the acid hydrobromate of quinine may be administered every four hours alternately with the alkali.This salt is extremely soluble,-ten grains to a drachm,-so that the dose can be administered ina single teaspoonful of water;and it has also the advantage of being less liable to produce nausea than the sulphate of quinine.

Antimony,aconite,and veratria are some of the useless or injurious,or even dangerous,medicaments which have from time to time been recommended for the treatment of rheumatic fever in children.Their action on the arthritic manifestations is uncertain,and,although the two latter have distinct antipyretic properties,their use in young rheumatic patients is negatived by the fact that they are,like antimony,depressant to the heart.

Gout responds satisfactorily to the exhibition of colchicum;rheumatic fever does not.Furthermore it is depressant to the heart,and its alleged ultility in certain published cases by no means warrants the exclusion of other and better agents from the therapy of the disease.

Iodide of potassium should not be given to rheumatic children,for there is evidence to show that it adds to the duration of the disease.

The old favourites,nitrate of potassium and lemon-juice are decidedly inferior to the remedies above advised,and the control of the malady can be better effected by other means.

I have never had occasion to administer antipyrine or antifebrine in these cases,though there is no doubt that certain observers speak highly of their action in cases of high temperature.As they have been known to produce syncopal attacks in grown-up persons,they had better be avoided in children's disease.

The action of carbolic acid and salicylic acid has been sought by the administration of salol,which is a compound of the two;it is without marked effects on the articular process and may produce the objectionable effects of the carbolic acid contained.

SYMPTOMATIC TREATMENT.

CARDIAC AFFECTIONS.

Forthwith on the occurrence of andocarditis or pericarditis, the alkaline treatment should be adopted, as giving the best results, salicylates or salicine being discontinued, and quinine substituted if the temperature is high. Warm poultices to the praecordia may be useful in endicarditis, but there seems to be no advantage gained by the application of blisters or leeches, as advocated by some. There is no connection between the circulation in the skin and that of the endocardium. With pericarditis it is different: there the superficial vessels have free communication with those of the parietal pericardium, and local depletion of the surface must directly relieve the hyperaemic of the serous membrane below. At the outset of pericarditis, one to three leeches, according to the age of the child, are often of distinct service. Care must be taken that the bleeding from the leech-bites does not go on too freely after their removal. Serious enfeeblement of the heart may occur from too great loss of blood through carelessness in this respect. Blisters, on the other hand, seem of chief value when there is effusion - not in the early stage of active inflammation. They cause children annoyance and distress, and should not be used unless strongly indicated in the absence of better means. A useful means of controlling pericarditis has been found in the ice-bag, particularly in the subacute recurrent pericarditic cases. These young patients usually bear it well, and even like it, and its power in relieving praecordial pain and tenderness and cardiac distress is very marked. It appears also to control the active inflammatory process, as shown by the speedy subsidence of friction and cardiac excitement. The ice-bag should not be too large and heavy. It should be applied almost continuously to the cardiac region, being removed from time to time if the effect is felt to be uncomfortably chilling or if the temperature falls below normal. Hot bottles should be applied to the feet and body, lest chilling should occur too readily, the effect of the cold application being carefully watched meanwhile. When, in cases of pericarditis or endocarditis, the heart is rapid and turbulent in its action, three to five drops of tincture of digitalis may be given every four hours to a child of five years for twelve or twenty-four hours, after which it should be given less frequently. Yet it must be administered cautiously; for it is a dangerous remedy when there is much pericardial effusion, or if the heart is greatly embarrassed by thickened adherent pericardium. When the palpitation is due to feebleness or dilatation, digitalis has great power to give force and tone to the cardiac contractions. Although stimulants should be avoided if possible, they are sometimes necessary when signs of heart-failure occur. In such cases they may be given freely to the amount of three ounces of wine and one and a half ounces of brandy in the twenty-four hours. Alcohol is wonderfully well borne by children; and it appears to act as a sedative and produce little or no excitement. It is superior to all other sedatives, even opium sometimes, in the cases mentioned. In practically all forms and stages of rheumatic fever opium is a remedy of immense value. There is no other drug which has the same power to soothe the urgent distress and

dyspnoea of dilatation of the heart, or the vomiting and pain of advanced pericardial disease, or the harassing cough which results from congestion of the base of the lungs, often preventing sleep, towards the close of valvular disease. To be effective it must be given freely, in doses of from a half to one minim to five or even ten minims of nepenthe or its equivalent every four hours, the dose being graduated according to the age of the child, the amount of distress, and the effect produced. The drug not infrequently not only relieves suffering and promotes euthanasia, but occasionally, by the ease and sleep it brings, gives the patient a further lease of life for a short time. The presence of pneumonia or bronchitis would contraindicate its employment. The vomiting which sometimes occurs at the close of pericarditis should be combated by ice, by small doses of dilute hydrocyanic acid and soda, and by the substitution of nutrient enemata for twelve hours for food by the mouth.

NERVOUS AFFECTIONS.

Should chorea be severe and the patient unable perhaps to sleep on account of the constant movement, chloral hydrate and bromide of potassium may be given in doses of five grains of each in syrup of orange every four hours until relief is obtained and the patient inclined to repose. Other nervous phenomena may be treated according to general principles and on the lines already enunciated in dealing with the adult disease.

BLOOD.

We have seen that anaemia is one of the characteristics of rheumatic feber in children; when the temperature has fallen and remained normal for seven days iron may be administered for its relief. The citrate of iron, in doses of five to six grains with ten to fifteen grains of soda and syrup of ginger in half an ounce of water, should be given as a precuation against relapse, especially if there has been any cardiac inflammation; or citrate of iron and quinine five grains with citrate of soda or potash in the same way, with two drachms each of water and chloroform-water; but in this case five grains of citric acid or a teaspoonful of lemon-juice must be added, to prevent the precipitation of the quinine by the excess of alkali which is practically always present in the soda or potash salt. If the anaemia is extreme, or the chronic symptoms persist, arsenic should be given with iron twice a day after food. This is the most efficaious of all drugs in the restoration of the blood-corpuscles; it should not, however, be prescribed until all symptoms of active inflammation are over, for it excites hyperaemia in the skin and mucous membranes, as evidenced by the reddened conjunctiva and tongue and flushed skin produced by full doses of the drug, and may presumably affect fibrous structures and serous membranes in the same way. The iron-arsenic combination may be effected by the administration of two drops of liquor potassii arsenitis with two drachms of iron wine twice a day after meals.

SKIN.

The erythema often observed in this disease usually vanishes of its own accord with the other morbid phenomena. No special treatment is therefore required.

TONSILS.

Tonsillar inflammation readily responds to

to the exhibition of the salicylates.

MANAGEMENT OF CONVALESCENCE.

The patient should remain in bed for at least ten days after all the acute symptoms have vanished, so as to insure against chill and preserve the quietness and integrity of the heart. Then tonic may be administered, and the rules recognised as suitable for the convalescence from all acute diseases brought into operation.

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C H R O N I C R H E U M A T I S M.

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S Y N O N Y M S.

Chronic Articular Rheumatism; Chronic Rheumatic Arthritis; Arthritis Rheumatica Chronica; Polyarthritis Synovialis Chronica(Heuter); Rheumatismus Articulorum Chronicus; Rheumarthritis Chronica; Rheumatisme Chronique Simple(Besnier).

D E F I N I T I O N.

Chronic rheumatism is an affection characterised by the occurrence of gradual and permanent changes in the articular structures,producing more or less deformity,and chiefly of the nature of a fibrous thickening and contraction.

It is much more rare in children than in adults, and is to be distinguished from the relapsing forms of acute rheumatism,where fresh exacerbations of a mild character, sometimes not more than stiffness and vague pains without swelling, recur from time to time. It usually begins quite insidiously as a chronic disorder, but may in rare instances result from an attack of the acute or subacute variety of the disease.The term, rheumatism,has been loosely applied to not a few affections of a nonrheumatic character,almost any obscure and obstinate pain which is not referable to some other agency being sometimes attributed to it.Chronic rheumatism properly so-called is a milder form of the subacute variety in whichthere is not sufficient local inflammation to keep the patient in bed or to give rise to pyrexial manifestations.In the same way that the acute merges into the subacute,so the latter may run into the chronic by insensible gradations.It is sometimes the precursor,not infrequently,some affirm,of an acute or subacute attack.It also exists independently of either.In it obstinate and sometimes shifting pains affect the articulations,muscles,and fibrous aponeuroses.The affected parts may be somewhat tender to the touch,but are not,as a rule,distinctly dwollen. The pain is increased by damp and cold.It often disappears in fine and returns in wet weather.Unless the patient is in easy circumstances he may never be confined to the house,and never consult a medical man,but may attend to business as usual until he is better,or until the onset of an acute or subacute attack compels him to

desist.It is a troublesome affection which may last for months,during which time there is not infrequently laid the foundation for future cardiac complications.

ETIOLOGY.

Chronic rheumatism may be the direct result of a single attack or more commonly of several attacks,of acute,or more especially of subacute,articular rheumatism. But it is generally a primary affection,occurring in persons who have not had either acute or subacute articular rheumatism,yet owning the same causation as these,and occasionally in its course exhibiting acute or subacute symptoms. The special predisposing causes are inheritance;repeated attacks of subacute or acute articular rheumatism,which in accordance with general laws impair the resisting-power of the affected joints; prolonged residence or employment in cold,damp,or wet rooms or localities;repeated exposure to bleak,cold currents of air or to frequent wettingsof the body or lower limbs.For these reasons it is most common amongst the poor,who are especially exposed to the influences just mentioned;and amongst them cellar-men and sailors, washerwomen and domestic servants,are very liable to the disease. It is chiefly an affection of advanced life,or at least of middle-age,and is rare in youth.The first attacks,and especially exacerbations,are apt to be induced by the direct action of a draught of cold air or by unusual exposure to cold and dampt air,especially when the body has been fatigued or overheated.In not a few instances there is no exciting cause discoverable.

PATHOLOGY.

The pathology of chronic rheumatism is not known to differ in any respect from that of the subacute and acute types already described.It possibly concerns an attenuated toxin or some obscute change in the fibrous structures,reducing their functional vigour,and rendering them less acutely susceptible to the excitant of the rheumatic taint.

MORBID ANATOMY.

The gross lesions of chronic rheumatism are not prominent,and they will vary with the severity and duration of the disease.The alterations are such as

chronic inflammation of a non-suppurative character might be expected to produce in the joints by one who had learned those characteristic of acute articular rheumatism.In the simple chronic form the proliferating process involves chiefly the synovial membrane,the capsular and other ligaments,and the periarticular tissues; to a less degree the cartilages,and to a much less degree,and exceptionally,the osseous surfaces.The synovial membrane is thickened,slightly injected,and its fringes hypertrophied and more vacular than normally.Little fluid usually exists in the joint unless during an exacerbation,when a moderate amount of thin,cloudy serum may be present;generally only a trace of thick,turbid fluid,containing oil-globules,and in severe cases débris of the cartilages,but no pus,is found.The fibrous capsule and ligaments become thickened,dense,and stiffened by hyperplasia;and sometimes the adjacent tendons and their sheaths,the fasciae and aponeuroses,undergo similar alterations,so that the movements of the joints become seriously interfered with.In some cases this irritative hyperplasia specially involves these periarticular structures,and these,undergoing retraction,produce marked deviations,subluxations,and deformities of the articulations very like those observed in rheumatoid arthritis,although the osseous components of the joints are unaffected.Jaccound gave to such cases the title of chronic fibrous rheumatism(Clin.Méd.de la Charité,Paris, 1867,Lecture 23).It is worth noting that Jaccoud's, Charcot's(Besnier,- Dict.Ency.,T.iv,p.680 et seq.),and Rinquet's(On Chronic Articular Rheumatism, Thèse de Paris,1879,pp.28-33)cases of so-called chronic fibrous rheumatism developed out of acute articular rheumatism, while Besnier's was primarily chronic.In simple chronic rheumatism,if protracted,the cartilages also proliferate, lose their **semitransparency** and polish,and become opaque and white;they are often rough and traversed by fissures, and occasionally present erosions;and these erosions are either naked or covered with a layer of newly-formed connective tissue,which may occasionally produce fibrous adhesions between the articular surfaces.Points of calcification occur in the cartilages and tendons in very chronic cases.Instances are observed in which the bones exhibit,to a slight degree,the alterations found in rheumatoid arthritis,and are probably transitional between the two affections.The muscles which move the affected articulations in severe cases are often atrophied,and the wasting imparts to the joints an appearance of considerable enlargement.Like rheumatoid arthritis, but unlike chronic gout,there is a marked absence of lesions arising from the arthritides,and the tendency to cardiac inflammations,so strong in rheumatic fever,is very slight in the chronic form of the disease.

SYMPTOMATOLOGY.

The symptoms are all derived from the condition of the joints and the phenomena to which this gives rise,and in simple chronic rheumatism many varieties

arise. In milder forms the patient experiences trifling or severe pain in one, or less frequently in two or more, joints, more especially in the knee or shoulder, or both, attended with a want of power in the member or with stiffness in the affected articulations. The pain frequently is likewise felt in the soft parts, muscular and tendinous, near the joints, and is usually increased by active or passive movement; it is not always accompanied by tenderness, and rarely with local elevation of temperature or swelling. The wearying aching in the joint is of an abiding character, but is very liable to exacerbations, especially at night; and these come on just before atmospheric changes, such as a considerable fall of temperature, the approach of rain, variations in the direction of the wind. etc., and they usually continue as long as the weather remains cold and wet. A very common symptom is a creaking or a grating which may be felt and heard during the movements of the joint. These symptoms may rarely prove more or less constant by night and day for years, but far more frequently, at least at first, they last an indefinite period and disappear to recur again and again, especially in the cold and changeable seasons of the year. Although in the earlier attacks, and often for a long time, no alteration of structure is perceptible in the painful joints, yet in some instances slight effusion into the articulation may be observed during the exacerbations, or the capsule and ligaments may at length become slightly thickened, or the muscles may waste and produce an apparent enlargement of the joint; and this prominence of the articular surfaces may be increased by retraction of the tendons and aponeuroses - a condition which causes real deformities, such as deviations and luxations, of the articulation and impairs more or less its movements. In very chronic cases a fibrous ankylosis may be observed. These last-mentioned conditions not infrequently give rise to great and long-continued suffering, and may even cause some anaemia and general debility; but very frequently the general health and vigour continue good, notwithstanding the permanent impairment of the functions of one or several of the large articulations, and the liability to exacerbations often amounting to attacks of subacute rheumatism from changes in the weather, exposure, or fatigue. In addition to these varieties there may be noted here a not infrequent one consisting of a series of attacks of subacute articular rheumatism recurring at short intervals, involving the same joints, and attended with slight elevation of temperature, febrile urine, perspiration, and moderate local evidences of synovitis, heat, pain, tenderness, swelling, and effusion into the affected joints. This is an obstinate variety, and is often associated with rheumatic pain in the muscles and fibrous tissues of the affected member. Simple chronic articular rheumatism, like the acute form, is most apt to affect the larger articulations, knees, shoulders, etc., but it frequently also involves the smaller ones of the hands and feet. Although usually polyarticular, it is prone to become fixed in a single joint, but even then it may attack several other articulations, and may migrate from one to another without damaging any. The course of the disease is usually one of deterioration during persistent or recurring attacks, and in many cases the intervals of relief become shorter and less marked; the joints become

weaker and stiffer;and although the pain may not increase and the general health may not be seriously impaired,yet the patients may continue for many years or the rest of their lives severe sufferers,unable to work,and often hardly able to walk even with the aid of a stick. Occasionally,after several years of pain and weakness, a sudden or slow improvement may set in and the patient become free from pain and lameness,and only experience some stiffness in the movements of the joints after several hours of rest,and slight thickening of the ligaments and capsule of one of more articulation. There is little danger to life,and the duration of the affection exhibits the utmost variation.

COMPLICATIONS.

The complications of the simple chronic affection are considered by not a few writers,and especially by those who regard the malady as constitutional or diathetic,to be the same as those of the acute form,and that they may precede,follow,alternate,or occur simultaneously with the articular affection.All admit that they are observed much less frequently in the former than in the latter.Others either deny the occurrence of visceral complications or do not mention them at all.Itis not denied that cardiac disease may be found in chronic articular rheumatism which has succeeded the acute form, and which may then be referred to the acute attack. The tissue changes than set up may not have produced at the time murmurs indicative of endocarditis,but these tissue changes may have ultimately roughened the endicardium, puckered a valve,or shortened its cords,so that cases of chronic articular rheumatism having a history of an acute attack cannot be safely included when inquiring into the influence of the chronic form upon the heart or other internal organ.Attention has not been sifficiently given to ascertain the frequency of the occurrence of these complications in primary chronic articular rheumatism, and reliable evidence is scanty. It is unlikely that the chronic form may slowly develop cardiac changes,as the acute form rapidly does;but when the advanced age of the persons most liable to chronic rheumatism is borne in mind,it must be admitted that valvular and arterial lesions,endarteritis,are observed at such periods of life independently of rheumatism,and rererable to such causes as repeated muscular effort, strain,chronic nephritis,senile degeneration.Somewhat similar observations are applicable to the attacks of asthma,of subacute bronchitis,of neuralgia,and of dyspepsia,which are frequently complained of by sufferers from simpe chronic articular rheumatism.Such affections are common in elderly persons in cold and damp climates; they may be mere complications rather than manifestations of rheumatism,or outcomes of the confinement and its attendant evils incident to chronic articular rheumatism,as is probably the relationship of the dyspepsia. The frequent coexistence of muscular rheumatism with

this variety is undoubted.

CHRONIC RHEUMATISM IN CHILDREN.

Chronic rheumatism is seldom encountered in the case of children, as compared with adults. It is to be distinguished from the relapsing form of acute rheumatism, where fresh exacerbations of a mild kind, sometimes nothing more than stiffness and vague pains without swelling, recur from time to time. But in certain cases affections of the joints, such as effusions or ankylosis, so remain in chronic form after an acute attack. Sometimes exostoses and bony formations in the muscles and tendons are liable to be developed in rheumatic children, occasionally to such a degree as to convert a large part of the muscular and tendinous system into a bony mass, rendering almost every movement of the body almost impossible (Henoch, - Diseases of Children).

D I A G N O S I S.

GENERAL CONSIDERATIONS.

There is seldom any difficulty, and usually none in recognising a case of chronic rheumatism, more particularly in the numerous instances in which it is left as a residue of rheumatic fever. When there is any difficulty, we may depend for a correct diagnosis chiefly upon the exclusion of all other causes but those of an atmospheric kind, capable of setting up chronic inflammation of the joints - one or more. If we take into account the patient's history, age, and occupation, the effect of the weather upon his symptoms, the fluctuating course of the malady, the absence of suppuration, etc., we can hardly fall into the error of confounding chronic articular rheumatism with traumatic, scrofulous, and other affections of the articulations.

DIFFERENTIAL DIAGNOSIS.

Arthritis Deformans.

The diagnosis of chronic rheumatism from arthritis deformans is sometimes to the superficial observer by no means easy; but the differentiation may be effected by taking into account the way it often grows out of an attack of acute rheumatism, by its being usually limited to one or, at most, to a very few joints, by its unsymmetrical character, and by its not causing the characteristic deformities of rheumatoid arthritis; but before the deformities have had time to develop it may be impossible to distinguish the two affections. It may be, too, that chronic articular rheumatism is capable of passing into arthritis deformans, the slow inflammatory process leading on to those structural alterations which occasion the characteristic deformities of the latter disease, under certain circumstances. It is also useful to bear in mind the fact that in arthritis deformans there is a progressive course, and also an absence of cardiac complications.

Articular Neuroses.

Here, in addition to the above-mentioned points, we have to remember, in distinguishing chronic

articular rheumatism from the articular neuroses,that, in the latter group of disorders,the subjective symptoms(pain,etc.)are strikingly disproportionate to the objective phenomena presented by the affected joint. Besides,the cutaneous sensibility is in an abnormal condition,hyperaesthesia,anaesthesia,etc., being observed.

Dupuytren's Contraction.

Careful examination of the affected joints,in connection with the history of the case and its progress,are of importance in differentiation chronic rheumatism from the above and old injuries in connection with the palmar fascia;furthermore,the deformities of the fingers induced are not of a symmetrical character.

Chronic Articular Gout.

It is often not easy to differentiate chronic rheumatism from chronic articular gout.Both are apt to be asymmetrical indistribution,to have paroxysmal exacerbations,to recur frequently without damaging the articulations,to have been preceded by acute attacks of their respective affections,and to be uncomplicated by endocarditis or pericarditis.But chronic articular rheumatism has no special tendency to attack the big tow;it is more persistent than gouty arthritis;it does not,even when of long standing,produce the peculiar deformities of the articulation or the visible chalk-like deposits in the ears or fingers observed in chronic gout.The etiology of the two diseases is dissimilar. There is no special liability to interstitial nephritis in articular rheumatism,which latter does not show the presence of urate of soda in the blood.

Tubercular Arthritis.

In chronic strumous or tubercular disease of a joint the youth,the personal and family history,and sometimes the evident defective nutrition of the patient;the moderate degree of local pain compared with the considerable progressive and uniform enlargement of the joint;the evident marked thickening of the synovial membrane,either early or lare according as the disease has originated in the synovial membrane or in the bones; the continuous course,without marked remissions or exacerbations,of the disease;the rarity with which more than one joint is affected;and the tendency to suppuration,ulceration,marked deformity,and final destruction of the articulation,- will prevent the disease from being mistaken for chronic rheumatism.

PROGNOSIS.

The prognosis,so far as life is concerned,is very favourable;but,as regards the prospect of complete recovery,it is just the opposite.In the majority of instances we must be content with affording the patient temporary relief fromsuffering:it is only in comparatively recent cases,when the patient is able to withdraw himself from all infurious conditions,that a more or less lasting degree of improvement can be maintained. By interfering with the nutritive processes,the effects

resulting from loss of sleep, due to pain, and inability to take exercise, the duration of life may be shortened.

TREATMENT.

PROPHYLAXIS.

The etiology of chronic rheumatism suggests an obvious line of prophylaxis, and the conditions obtaining are much the same as in the acute form of the disease. Individuals who have already suffered from the latter affection should be advised, in order to ward off chronic articular rheumatism altogether, or its exacerbations when it is already established, to live in a dry-warm house, and, when they are existing under unfavourable climatic conditions, to seek, either for a time or permanently, a residence elsewhere.

GENERAL MANAGEMENT.

Hygienic treatment constitutes an essential, if not the most valuable, part of the curative and palliative management of chronic rheumatism. It matters little what diet the patient has, so that it is ample, nutritious, easily digestible. It should comprise chiefly meats, fresh vegetables, and cereals. Sweets, pastry, sweet wines, and malt liquors should be avoided. Claret or well-diluted whisky may be allowed. Tea and coffe may be taken. The patient should adopt and continue moderately active exercise until compelled to omit it on account of the advancing lesions of the joints. The wearing of flannel next the skin, sufficient clothing, and residence in a warm and dry house, etc., are points of great importance. In fact, all the known or suspected causes of the disease should be removed as far as possible.

MEDICINAL TREATMENT.

There is no drug which exerts an absolutely curative influence on chronic rheumatism. Although, like everything also in chronic articular rheumatism, it sometimes fails, there is perhaps no single remedy which afford so much relief to the pain and stiffness of the joints as sodium salicylate. Numerous cases recorded in the literature testify to this, amongst them those of Jacob (Brit. Med. Jour., 1879, ii, 171), who reports some benefit in seventy-five per cent. out of eighty patients treated by this drug. It must be given in full doses, and be persevered with. Salicylate of quinine should be tried if there be much debility or if the sodium salt fail. Propylamine or trimethylamine is deserving of further trial in this disease. From one to two hundred grains may be administered in peppermint-water in the twenty-four hours. Iodide of potassium, cod-liver oil, arsenic, iodide of iron, and quinine are all and several remedies from which more or less benefit is derived in chronic articular rheumatism. The combination of iodide of potassium with guaiacum resin, two to three grains of each three times a day in syrup and cinnamon-water, is sometimes very useful. Some writers have seen success attend the exhibition of bromide of lithium. When the skin is habitually dry and harsh, a dose of pilocarpine, every

other night for a few times,will often give pleasing results.I have employed piperazine internally,in doses of fifteen grains,but the effects observed were not such as to warrant my continuing it.In some cases the simple alkalies,or a course of alkaline waters,may prove beneficial,and debilitated patients need tonics,of which the already-mentioned cod-liver oil and arsenic are the most suitable for the disease.

HYDROTHERAPY.

As a rule,hydrotherapeutic measures are of great servive,as adjuvants,in the treatment of chronic articular rheumatism,for which warm baths,both local and especially general,enjoy a very high reputation.In relieving pain and causing the structural changes to subside their value does not depend,either wholly or chiefly,on their chemical composition.Prolonged immersion in plain water at a temperature of from ninety-five to ninety-nine degrees,or even higher,is of the most beneficial.We may safely ascribe the good effect,at least in part,to the influence of the bath on the nutritive processes,especially ny exciting secretion,and to their topical action in dilating the blood-vessels,and thus modifying the circulation through the affected part,and saturating the tissues with moisture.These effects may be supplemented by many others,-e.g.,the production of electrical currents,-which,though individually trifling, may aid one another by their simultaneous and concerted action.Whatever constituents the baths may contain besides hot water may broadly be said to act by stimulating the sensory nerve-endings in the skin,either momentarily, or,when the body is immersed for a long time in water containing salts which impregnate the skin,during a longer period of time.This stimulation exercises an influence on the functions of circulation and respiration, and also perhaps on that of secretion.The precise nature of the irritant,whether it consists of salts or gases in solution,such as carbonic acid and sulphuretted hydrogen, is of little moment,providing its influence in causing absorption is increased by friction of the affected joints. Such baths are,however,often ill borne by weakly or excitable subjects.For the rest,in choosing such additions to artificial baths,and in our choice of natural thermic springs,we are guided less by any belief in the specific virtue of one or other of their constituents than by the accidental or collateral conditions. The only feature that these natural springs whose reputation is of long standing,and which really have a healing power,have in common is their high temperature;they differ widely in their chemical composition,belonging either to the group of indifferent thermal waters,or of sulphuretted,saline,or alkaline waters.Of the various thermal baths there are:aerothermal baths,sodium chloride baths,brine baths,peat baths,and mud baths.These baths have in common the property in common of exerting a favourble influence upon the resolution of the structural alterations induced by the rheumatic process,and absorption of the effusions,by stimulating metabolism, by increasing the secretory activity of the skin,and by locally inducing changes in the affected parts through dilatation of the vessels,by influencing the circulatory conditions,and by irrigating the tissues;in connectionwith which,besides,the high temperature of

the bath neutralises the increased sensitiveness and irritability of the diseased parts. Nevertheless, the stimulation induced by the baths at various places is different, and permits of a selection in accordance with the needs of the individual case. The action of the hot peat baths and mud baths is the most intense in the larger number of cases of chronic rheumatic affections, whether involving the muscles, the fasciae, the periosteum, the synovial capsule, and the ligamentous structures, or other fibrous tissues. These baths contain, as stated, in greatest abundance, salts, gases, and other cutaneous irritants, and are capable of exerting pressure and friction mechanically; besides, they can be employed at higher temperatures than mineral water baths. Peat baths and mud baths, which combine the effects of baths and of massage, favour, often to a surprising degree, the absorption of fluid effusions, and the retrogressive metamorphosis of inflammatory products of some standing. In even unfavourable cases of rheumatic palsy they at least counteract the rapid atrophy of the paralysed muscles, and in less favourable cases they exert a useful influence as they restore nerve conduction, which has been deranged by the presence of exudates. As a preliminary procedure, and during the after-treatment, in the various forms of rheumatic ankylosis, a course of peat baths and mud baths is of service. Among antirheumatic baths of this character the highest reputation is enjoyed by the peat baths of Marienbad, Franzensbad, Neudorf, Elster, and Muskau, and by the sulphurous and mud baths of Pistyan, Trencsin, Warasfin, Nenndorf, and Eilsen. The acrothermal waters of Teoplitz-Schoenau, Warmbrunn, Ragatz, Plombières, Daruvar, Topusko, Toeplitz-Krapina, and others; and in the United States of America, Hot Springs, in Arkansas, and Lakewood, in New Jersey, are most useful, especially for the chronic rheumatic affections due to cold, and their consequences, as well as in theprresence of marked hypersensitiveness and a tendency to rheumatic relapses. Such thermal baths as are not situated too high in altitude, but enjoy a sheltered climate, shielded from sharp winds, and with not too wide variations in the daily temperature, are deserving of consideration. In the United States suitable waters are Hot Springs, in Arkansas, Hot Springs, in Virginia, Mount Clemens Springs, in Michigan, and Minnekahta Spring, in South Dakota. The favourite baths in Germany and Switzerland are: Pfaeffers and Ragatz, Gastein, Wildbad, Roemerbad, Leuk, Teplitz, Warmbrunn, Baden (near Vienna), Baden (in the valley of the Aar), Baden-Baden, Landeck, Schinznach, Aix-la-Chapelle, Wiesbaden, and other hot brine springs, - Nauheim, Oeynhausen; in France: Plombières, Aix-les-Bains, Barèges, Bagnèresde Luchon, Nèris, Luxeuil, Mont-Dore, etc.; in Hungary, the highly effective Hercules baths in Mehadia, Trentschin, etc. It need scarcely be pointed out that the treatment by local baths must be restricted to a few only of the joints: the fact is obvious. They may serve to assist the action of the general baths in promoting absorption; or they may be employed when, for any reason, the use of general baths is inadmissible: when, for example, there exists some valvular lesion of the heart, possibly bequeathed by a previous attack of polyarthritis; or when the vessels are diseased; or, in short, when there is any condition that forbids the employment of a high temperature and with much gas

in them.In chronic articular rheumatism,when valvular disease of the heart exists as a complication,the thermal brine baths of Nauheim,Rehme,and Kissengen deserve the preference.The operation of loca,like that of general baths,may be assisted by adding various stimulating ingredients to the water,especially peat and mud;and their temperature,owing to the limited extent of the surface exposed to them,may be higher than that of baths in which the whole body is immersed,thereby increasing the activity of the local perspiration and absorption.Of great service in cases of long standing are hot sand baths,ata temperature of from 112.5.to 131.F.,in duration varying from a half to an hour and a half;owing to the ease with which they may be procured,they are especially suited to patients in humble circumstances.For home use in the treatment of chronic rheumatism pine-needle baths,prepared by the addition of broken branches of pine,or of pine-needles,to the baths,at a temperature of 97.to 104.F.,may be employed, followed by rest in bed for several hours.Another alternative is to use poultices of hot fango - the volcanic mud exported from Italy. The waters are employed also for drinking purposes at some of the health resorts already mentioned(at the acratothermal springs,the sulphurous thermal springs,and the sodium chloride springs), in addition to the bathing cure,in order to thoroughly flush out the organism and maintain the diaphoresis induced by the bath.The essential object of vapour baths and of the methods employed in hydrotherapy,viz.,the wet pack,rubbing and drenching and douching with water, is likewise to cause perspiration,and thereby to promote the absorption of exuded matters;but such methods should be employed with caution,and reserved for the recent cases of the disease in strong and otherwise healthy persons;or we may employ them tentatively,when other curative measures have been tried and failed.The natural vapour baths should also be resorted to with caution;for,though they certainly induce copious perspiration,the disturbance of the circulation they bring about is apt to constitute a real danger.

LOCAL TREATMENT.

Local measures are deserving of more respect than internal medicaments;indeed,they should never be omitted. Their object is,on the one hand,to remove the inflammation and swelling of the joints,and to alleviate pain or other annoyance,on the other.Especially of resorted to early,local treatment will often produce a decided improvement,or check the further progress of the disease.The patient must be oonvinced that any such treatment must be persevered with,as fully a month may elapse befofe such decided results are obtained.Hydrotherapy yields the best results,and one often sees great benefit arise from the local hot-air or hygrothermic treatment.The usual course consists in applying a stream of heated air(by a lamp to 240. to 280.F.)for forty-five to sixty minutes,the hand or knee being suspended in a box which has been adapted to fit any joint by means of adjustable sides.Strong local perspiration is induced,with dilatation of the peripheral vessels and improvement in the activity of the loca circulation,which promotes absorption.Great relief from pain and stiffness is often experienced,especially if

the application is followed by Swedish movements.Alternate douching with cold and very hot water,or wrapping the joints in flannels wrung out in steaming water,acts in a similar manner.If the knees are involved,occasional cantharides blisters,or the thermo-cautery should be applied,but in elderly or debilitated subjects care should be taken not to irritate the skin too much.The wearing of woollen kneecaps and gloves gives comfort,as does the packing of the swollen joints in cottonwool and oilsilk.Galvanism is of doubtful value,but general massage is useful.One sometimes derives benefit from encasing the joints for several hours in a two per cent. solution of piperazine in water.Other topical applications to relieve pain are tincture of iodine,iodine ointments,ichthyol ointment(forty grains to the ounce of lanoline),guaiacol and glycerine in equal parts,and the salicylate of methyl.

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M U S C U L A R R H E U M A T I S M.

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S Y N O N Y M S.

Myalgia; Myalgia Rheumatica; Myopathia; Rheumatisme Musculaire; Muskelrheumatismus.

D E F I N I T I O N.

Muscular rheumatism,or myalgia,is that variety of rheumatism in which the symptoms are localised in various muscles,with usually little,if any,constitutional disturbance.Various names,more or less descriptive of the part of the body affected,are applied to this disease - lumbago,torticollis,and pleurodynia,for example.

Consequently,the affections included under this term are certain painful disorders of the fibro-muscular structures.They are commonly found in persons the subject of the rheumatic diathesis,and are characterised by pain and often spasm,and sometimes a slight degree of fever.No doubt as our knowledge increases so many many attacks connected with painful states of muscles and fasciae are eliminated from the somewhat uncertain group of muscular rheumatism.True inflammation is not believed to exist,and pathological investigation has rarely shown any morbid changes in the affected parts. The symptoms,therefore,have been attributed to some temporary hyperaemia,slight serous exudation,or neuralgic state of the sensory nerve-filaments.The strongest support is given to this statement from the absence of any marked tenderness in such affected muscles as can be sufficiently examined.In certain cases,undistinguishable clinically,it is quite probable that a periarthritis affecting a joint is in reality the principal factor in the case.In others,again,a subacute rheumatism affecting a joint seems to spread to the adjoining tendon sheaths,and this secondarily to attack the muscles themselves,the affection of which may ultimately remain the only condition present.That many forms of muscular rheumatism are indeed of nervous origin rather than dependent upon inflammatory changes in the muscular or interstitial connective tissue,is proved by cases in which the superficial muscles,as already mentioned, are accessible to examination,e.g.,the sterno-mastoid, and are seen to be involved.We find the muscle spasmodically contracted,but not,as a rule,more painful than and uninflamed muscle in a state of tonic spasm;more-

over,the muscle is not nearly so tender to the touch as it would be if it were really in a state of inflammation, pain being caused by forcible attempts at extension,and the case appears to be one of simple tonic spasm.

E T I O L O G Y.

The idea is fast gaining ground that muscular rheumatism is a disease of infectious origin,and due to a germ. It is probable that in the near futute the truth of this affirmation will be definitely established.

All ages are liable to its occurrence,but the part affected varies with the time of life,children and young adults being much more subject to torticollis,and older individuals to lumbago and general rheumatism of the limbs.

Amongst hospital patients the disease prevails more amongst men than women,owing doubtless to the greater exposure of the former to the cold;but amongst other classes the same difference is not seen.

It is usually observed in all countries,but,according to some writers,it is unusually frequent in tropical countries,although there acute rheumatism is very uncommon.

The other predisposing causes of muscular rheumatism are mainly exposure to cold and strain or fatigue of muscles.If these two conditions coexist - e.g.,standing in a draught of cold air or lying on the ground when fatigued - the chances of the affection coming on are greatly enhanced.Strain,a twist of the body,or a false step can actively start an attack of this kind,and by the sufferers themselves it is constantly attributed to this cause.The part played by this element is difficult to determine,a very slight strain being often followed by great pain and distress from the subsequent rheumatic affection.

Some individuals are specially prone to attacks,the slightest current of air,change of clothing,etc.,being sufficient to determine its occurrence.These persons are found to have suffered from rheumatism in some other form,and thus we must in them consider that the rheumatic diathesis furnishes the reason for their unusual susceptibility.

In gouty families muscular rheumatism has been observed to be common,and therefore it is reasonable to presume that a gouty disposition favours the occurrence of the disease under consideration.

According to some, heredity is one of the most potent factors in the predisposition.

Occupations involving exposure of various kinds are similarly operative,lumbago being often developed in common labourers who have strained their backs in lifting or digging in damp,heavy soil.

One attack would seem to predispose to another.

The muscles affected are usually the larger ones

concerned in effort, such as the lumbar, deltoid, sterno-cleido-mastoid, rectus, scapular, pectoral, intercostal, etc., but almost any of the larger voluntary muscles may be attacked.

PATHOLOGY.

The general opinion nowadays is that the pathology of muscular rheumatism is identical with that of the acute variety of the general taint. Leube thinks that the affection may be epidemic at times, and that it is possibly due to an attenuated form of the toxin of the articular type of the malady.

MORBID ANATOMY.

The pathological anatomy of muscular rheumatism is still imperfectly understood, as the disease is init-self never fatal, and there is some doubt as to whether the lesions are to be found in the muscles themselves or their nerves. It is generally believed, however, that there is an acute inflammation affecting certain voluntary muscles, their tendon-sheaths, and fascia, and that portion of the neighbouring periosteum to which they may be attached. In fatal cases of acute articular rheumatism in which the muscles have been involved their fibres have exhibited swelling and granular degeneration with vacuoles. In severe cases decided muscular atrophy has been observed, indicating a tropho-neurosis, but this condition is rare as a result of simple myalgia. It may be prominently seen in the deltoid muscle when the shoulder-joint is involved. There may be round-celled infiltration, increased nuclei in the muscle-fibres, and increased connective tissue around the fasciculi in this disease.

SYMPTOMS.

An attack of muscular rheumatism is characterised by the occurrence of pain, and this in many cases is the only symptom observed. In all except the more aggravated attacks, pain is experienced only when the affected part is disturbed. In such, when complete rest or fixed immobility is maintained, there is comfort, or at most a somewhat dull and uneasy sensation; but when any contraction of the muscle in question is produced, whether voluntary or otherwise, severe and often excruciating

pain is at once experienced, often giving rise to a sudden cry or causing the features to be contracted in a grimace. The suffering ceases almost at once when the muscular contraction is relaxed. In more aggravated attacks the more is more severe, and persists besides, though to a less degree, even when there is no contraction. In rare instances, when the maximum degree has been attained, there is continuous pain; but the affected muscles are persistently maintained in a relaxed condition by means of true spasm of the surrounding muscles. Slow passive movement affects the subject of muscular rheumatism, and may often be accomplished by a little management without causing pain. If, at the same time, these muscles be handled by pinching and slight pressure, it will be found that they are very sensitive to the touch. When some tenderness does exist, it is slight and is not located in the district of the lower nerve-trunks. Pressure even sometimes allays pain. The constant effort to avoid pain gives rise to a feeling and appearance of stiffness, and this characteristic attitudes and positions of the head, trunk, or limbs are voluntarily and persistently maintained. There is no spasm of the affected muscles; the distortion is the result of stiff contraction of the associated muscles, which thus forcibly fix the faulty one and hold it in a state of relaxation. Cramp or spasmodic contraction of a single muscle of a painful character does, however, sometimes occur in rheumatism sufferers, and much resembles the condition above described. In the same persons also muscular rheumatism may occur in a much more fugitive or erratic form, frequently being nothing more than a slightly painful condition of some group of muscles which have in some way been exposed to cold. This may last but a short time, and either spontaneously disappear or be readily removed by exercise or friction. Muscular rheumatism is generally confined to one muscle or a single group of muscles. Those most liable to it are the very superficial and those easily exposed to cold, such as the deltoid and trapezius, powerful muscles subjected to violent strain, the lumbar muscles for example, and those aiding in the formation of the parietes of large cavities. The disease very commonly exists without any constitutional disturbances, but sometimes there are present the symptoms of pyrexia, such as slight elevation of temperature and temporary disorder of the digestive organs, loss of appetite, constipation, and general malaise. The acute forms generally last but a few days, terminating by gradual subsidence and final disappearance of the pain. The fugitive kind, already alluded to, may, however, be present more or less during several weeks.

VARIETIES.

There are several varieties of muscular rheumatism, divided according to location, and these may be separately described as follows:

LUMBAGO.

Lumbago (Myalgia Lumbalis, Rheumatism of the Lumbar Muscles) is a very common form of muscular rheumatism, and there are probably few who have not, at some time or other, suffered from it. It attacks the lumbar muscles and the strong aponeurotic structures in connection with these. It is more frequently than any other form attributed to some effort of lifting or sudden

twist of the body,but in many cases it owes its origin directly to exposure to cold or damp,it being not uncommon for persons who have fallen asleep on damp ground to find that they are unable to get up,when they awake, owing to lumbago.It is much more common in males than females,doubtless because men are more exposed to its exciting causes.It is more prone to become chronic and to recur than any other form of muscular rheumatism.For this reason,and also on account of the annoyance and suffering it entails,it should be vigorously treated from the outset by the measures which will presently be described.The pain comes on suddenly and renders the person helpless,the body,if he is able to go about,being held stiffly to prevent any movement or bending;if severe,he is absolutely compelled to observe complete rest in bed.The muscles,when handled,appear slightly sore,but no local point of acute tenderness can be found.This fact,with the characteristic shrinking from any movement,distinguishes lumbago from neuralgia and from abscess.Pain in the loins,more or less severe,is such a frequent accompaniment of disorder of the sexual organs and parts that careful examination should always be instituted lest some serious organic disease with lumbar pain as a symptom be mistaken for a simple lumbago.Perinephritis,lumbar abscess,spinal disease,abdominal abscess,and disease of the uterus and rectum are the most important of these.

PLEURODYNIA.

Pleurodynia(Myalgia Pectoralis vel Intercostalis,Rheumatism of the Pectoral and Intercostal Muscles) is characterised by the occurrence of rheumatic trouble in connection with the intercostal and sometimes the pectoral muscles.Spasmodic pain is felt in one or other side of the chest,and is especially aggravated by the movements of respiration;it is rendered intense by the efforts of coughing or sneezing.Pleurodynia may be confounded with pleurisy,the distinguishing features being the absence of fever and the friction sound of pleurisy. Intercostal neuralgia is sometimes with difficulty known from pleurodynia,but in the former the pain is more circumscribed,more paroxysmal,and more easily aggravated by pressure than in pleurodynia,and when severe there are tender points in the course of the nerve a little outside of the middle line posteriorly - dorsal point and anteriorly - sternal point.Now and then the hyperaesthetic areas become anaesthetic,and even patches of herpes may form in the course of the nerve,when doubt can no longer remain.From periostitis of a rib pleurodynia may be known by the fact that in the one the tenderness is marked in the intercostal space,and in the other in the rib itself.Pleurodynia is a frequent accompaniment of thoracic affections,causing cough, the frequent paroxysms of coughing tending to induce a painful state of the overworked muscles.The pain,which may be very great,can often be controlled by fixing the chest with imbriacted plaster or a firm bandage,etc.

TORTICOLLIS.

Torticollis(Mylagia Cervicalis,Torticollis Rheumatica,Cervicodynia,Caput Obstipum,Stiff Neck,Wry Neck)is an affection including those cases of rheumatic idiopathic troubles of one or more of the muscles of the side and shape of the neck,which fixes the head firmly in the median line or else in a twisted fashion,

with the face towards the sound side.The disease can be recognised at a glance by the peculiar manner in which a person will turn his whole body round instead of rotating his head alone.It is much more common in children than in adults.The sterno-mastoid is the muscle chiefly affected,but any of the muscles of the neck may become rheumatic in the same way,and frequently several of them suffer at the same time.The most important point at the outset of an attack of wry neck is to determine whether we have to do with a true idiopathic rheumatic disorder, or whether the muscular stiffness is secondary to some spinal or vertebral lesion.The diagnosis is usually founded upon the suddenness of the onset,the absence of other symptoms of nervous disease,and the rapid course of the illness,terminating in cure in a few days.

SCAPULODYNIA.

Scapulodynia(Myalgia Scapularis,Omalgia)is an affection in whichthe muscles attached to the scapula and humerus are painful and rigid.

CEPHALODYNIA.

Cephalodynia(Myalgia Cephalica s. Capitis, Rheumatic Caphalalgia,Rheumatismus Epicranii,Rheumatism of the Head)is a rheumatic affection with localisation in the muscles of the occipital,frontal and temporal regions,and in the pericranium.Movements of the head, pressure of comb or hat,cause sensible and sometimes very severe pain.This affection may be easily mistaken for periostitis,hemicrania,or headache due to intracranial mischief;perhaps it is more correct to say that these disorders are frequently mistaken for rheumatism of the head.It is not until they have been all excluded, and the pain shown to be symmetrical and really localised in the muscles or the fibrous aponeurosis by the effect of movement in exaggerating it,that the diagnosis of rheumatic caphalalgia can be established.

ABDOMINAL RHEUMATISM.

This term is sometimes employed when the muscles of the abdomen are involved.

OTHER FORMS.

In addition to the muscular groups just mentioned,any muscle in the body is liable to be affected by rheumatism.Some authors,especially the older ones, go so far as to speak of rheumatism of organic muscles, as they term it,including the uterus,diaphragm,heart, intestines,bladder,etc.These affections are all characterised by a sinlgle feature,namely,pain,which is referred,with more or less show of justice,to the muscular tissue.There is no way of distinguishing them, when they occur in organic muscles,from such neuroses as colic and cardialgia and angina pectoris,or even from true inflammation;this is the less important as they all require the same treatment.Further,un der the name of vague muscular rheumatism,we are acquainted with wandering pains,now in one muscle,now in another, or in the tendons and fasciae;these pains may occur either with or without fever;they are termed vague muscular rheumatism when no cause for them can be assigned,or when they are preceded by a chill.They often assume a neuralgic character and may alternate with true neuralgia,such as of the fifth nerve.Sometimes after shifting from place to place,the pain settles in some particular group of muscles,or is limited to the

area of distribution of a particular nerve.

D I A G N O S I S.

We have already seen how important it is to exclude all other affections which may give rise to muscular pain from the diagnosis.Such pain is a very common symptom of many local and constitutional disorders at some period of their course,and especially before their characteristic signs are manifested;this accounts for the ease with which both lay and professional opinion may be misled into ascribing muscular pain and cramp to muscular rheumatism,when they really form part of some wholly different malady.Such rheumatoid pains - sensations of dragging and tearing in the muscles - are most frequently associated with organic disease of the vertebral column and spinal cord,notably tabes,which has peripheral pain as an early symptom;functional disorder of the same part,as hysteria or spinal irritation;intrathoracic inflammation;chronic poisoning by lead and mercury;and painful spasm from deep-seated inflammation or suppuration.Of acute affections the exanthemata,especially smallpox,and trichinosis - the former at their outset, the latter during its course - are prone to be attended by pain in the muscles,more or less generalised. Again, painful spasm of the muscles is often of reflex origin,caused by deeply located inflammatory processes or painful affections in the neighbourhood;some such mischief ought always to be suspected when spasm is associated with muscular pain.Finally,muscular rheumatism may be confounded with neuralgia,particularly when the symptoms of the latter are indefinite,when deep-seated nerves are involved,when the pain fails to correspond to the course of a particular nerve-trunk,and radiates over a wider area than usual.It is sufficient to indicate these various sources of fallacy,which,if remembered,can generally be guarded against by a consideration of the special features of each one of them.

P R O G N O S I S.

The vast majority of sufferers from muscular rheumatism recover in a few days,but cases of exceptional severity,or occurring in debilitated or highly rheumatic subjects,may prove intractable and linger for several months without the desired improvement and cure.

TREATMENT.

PROPHYLAXIS.

For the reason that one attack of muscular rheumatism predisposes to another, preventive treatment should consist of avoiding exposure to cold and wet, the sudden arrest of perspiration, muscular strain, fatigue, etc., everything essential to the maintenance of perfect nutrition and the proper quality of the blood being done. Such persons should wear woollen undergarments in winter, and keep the skin in good condition by bathing and massage; in short, the same measures of prophylaxis are to be observed as were advised for the prevention of chronic articular rheumatism. A Turkish bath, followed by brisk friction, may sometimes abort an expected attack.

MEDICINAL TREATMENT.

Drugs are not often required for the relief of this disease. Some acute cases are benefited by the salicylate of soda, and in very protracted cases the general tonics, such as arsenic, nux vomica, and cod-liver oil, are serviceable. Potassium iodide, sulphur, and guaiacum have all been extolled, but their value is questionable. The coal-tar products, antipyrine, phenacetine, etc., are usually disappointing. Good, nourishing mixed diet of plainly cooked food, with meat once or twice a day and plenty of fresh vegetables, is indicated in all cases. The action of the skin should be promoted to diaphoresis. For this purpose the hot-air or Turkish bath at the outset would, as stated, seem to be really abortive. The most reliable amongst the medicinal measures are acetate of potash and Dover's powder. Pilocarpine occasionally proves useful; citrate of potash, and other fixed alkaline salts, are also given. Benefit from the use of salicylate of soda will be in proportion as the evidence of the rheumatic constitution is well marked, as shown by the tendency on other occasions to attacks of acute articular rheumatism.

LOCAL TREATMENT.

The indications for treatment are to relieve the pain and to counteract the diathetic condition generally present. The relief of the pain is accomplished in various ways, according to the seat of the trouble. In severe cases it is proper to resort to the hypodermic use of morphia, to which may advantageously be added some atropine. When the pain is seated in large muscles, the injection will produce better results if thrown not merely under the skin, but into the substance of the muscle. Sometimes perfect rest in bed is necessary to secure the required immobility; in other cases this can better be secured by plaster or firm bandages. Soothing anodynes are extremely useful locally, and counter-irritants also may be used with benefit. Liniments give us a convenient form of application. The best are those containing a considerable proportion of chloroform, or turpentine or belladonna. A twenty per cent. ointment of salicylic acid sometimes gives relief. Among local applications none is sometimes so effectual as the Paquelin thermocautery, applied gently and rapidly over a large surface.

If the application is not too vigorous,it can be repeated several days in succession if need be,but its effect is often magical.Galvanism sometimes proves a rapid cure.The repeated application of tincture of iodine often gives relief,and aconite is a useful agent. Continuous heat is nearly always grateful,and may be applied either in the dry form or by means of soft warm linseed poultices with or without a percentage of mustard.When these are discontinued,care should be taken to protect the affected muscles by keeping them enveloped in woollen or flannel coverings.Hot water bottles are sometimes of signal service,and turpentine stupes also give relief.In obstinate cases cantharides blisters may be used,but hot-water douching is better.For lumbago acupuncture has been tried,and deep hypodermic injections of distilled water probably act in a similar manner as counterirritants.Common domestic remedies are porous plasters and ironing the muscles with a hot flat-iron through a thick piece of paper or flannel.Massage should be practised as the patient improves.

The TREATMENT OF THE VARIETIES is conducted on smilar lines.The pain in pleurodynia may be quickly relieved,as a rule,by dry or wet cupping,with warm applications to the surface,or by a subcutaneous injection of morphia.Should these means fail,we may resort to counterirritation by mustard poultices or blisters, or by the continuous current.There is nothing special in the treatment of torticollis beyond what has already been said under the general heading;and the same is true of the other forms.

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GONORRHOEAL RHEUMATISM.

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SYNONYMS.

Gonorrhoeal Arthritis; Gonorrhoeal Synovitis; Venereal Rheumatism; Blenorrhagic Rheumatism; Urethral Synovitis; Urethral Rheumatism; Gonocele; Arthrite; Arthropathie Blenorrhagique; Tripper-rheumatismus.

DEFINITION.

As implied by the name, the occurrence of this disease is due to gonorrhoea; it is an acute infectious ailment characterised by localised inflammation in one or more of the larger joints of the extremities, and due to the septic mischief induced by the gonococcus.

It is generally seen to develop at the close of an attack of gonorrhoea, but sometimes also during the acute stage of the disease, or at any period during the course of gleet.

Though it has apparently no essential connection with true rheumatism, its name demands that it should receive special consideration.

ETIOLOGY.

Although commonly and erroneously called gonorrhoeal rheumatism, this disease has no etiological relationship with acute articular rheumatism, being caused exclusively by the migration of the gonococcus or the transference of its irritating toxins from the urethra to the joint. In a number of instances the gonococcus itself has been demonstrated in the articulation; in other cases where it has not been found it may have been previously present and destroyed, or the germ products have been conveyed by the vessels from their site of development in the urethra to set up a fresh irritation in the fibrous structure of the articulation concerned. Consequently, the etiology of gonorrhoeal arthritis is that of gonorrhoeal urethritis. It is accordingly more prevalent in young unmarried males than in females. It may, however, be acquired by any one who has been infected with gonorrhoea. The heinous hypothesis held by certain of the lower orders, especially amongst

degraded and ignorant foreigners,that a man can rid himself of a dose of the"clap"by imparting it to a young child,results sometimes in infection in extreme youth. Thus,Chiasio(Gaz.Méd.di Torino,Feb.15,1894)and Isnardi of Turin have reported a case in a child of ten years, and Richardiere(L'Union Méd.,Oct.26,1893)of Paris reported cases in girls between five and ten years,and says that he has even known of its occurrence in a child before twelve months of age.

Conditions which influence the development of acute articular rheumatism,such as exposure to cold and wet,have little or nothing to do with gonorrhoeal arthritis,but injury to a particular joint may have some in-fluence in localising the inflammation there.Broadly speaking,while the ordinary exciting causes of simple acute articular rheumatism are not necessary to the production of this disease,they do not and then actas adjuvants.Such are cold,fatigue,and,as stated,injuries to the joints,and a severe acute arthritis is not infrequently developed during gonorrhoea under such circumstances. Other predisposing causes probably exist,the absence of which in some measure explains the infrequency of gonorrhoeal arthritis as compared with the prevalence of gonorrhoea.Besnier(loc.cit.)holds that constitutional rheumatism,the arthritis habit,or l'hérédité arthritique,is not infrequently present in the victims of gonorrhoea rheumatism as a predisposition; Nolen(Gonorrhoeal Rheumatism, Deut.Arch.f.klin.Med.,Bd. 33,1883)found an inherited rheumatic predisposition in six out of eighty-eight cases,and four others who had rheumatism before contracting gonorrhoea;and Hutchinson maintains that it is the existence of arthritic diathesis which enables urethral inflammation to produce gonorrhoeal arthritis.He says:"From statistics that I have carefully collected I have no hesitation in believing that the predisposing cause of it usually is the inheritance of arthritic tendencies;"and adds,"Very often the subject of gonorrhoeal rheumatism will give a family history of gout"However,the disease often occurs in the absence of any discoverable tendency,hereditary or acquired,to simle articular rheumatism.On the other hand,persons who have had one or several attacks of gonorrhoea previously that did not give rise to rheumatism. Nolen's table of eighty-eight cases contains twelve instances of this kind.It is held by some that,by reducing the resisting force of the organism,scrofula,the so-called lymphatic diathesis,anaemia,and debility favour the development of the affection.

Like the parent urethral disease,this affection is, proportionately as well as actually,much more frequent in men than in women,as instanced by Nolen's one hundred and eleven cases of the former to seven of the latter; and the greater proclivity of men has been attributed by certain writers to the greater delicacy,sensibility, and complexity of the textures invilved in them than in women by the gonorrhoeal infection.

Now and then writers of experience have affirmed that this type of arthritis may complicate a simple non-specific urethritis in the male,or even leucorrhoea or menstrual disorders in the female,but such cases must, I think,be very rare.Selle(Clinical Medicine,Berlin, 1781)and Swediaur(Lond.Med.Gaz.,1781),who were the

first to indicate the cause of thedisease,also pointed out that an affection apparently identical is in very exceptional instances seen to be associated with non-contagious urethral discharge,and with the urethral irritation incident to catheterism and to stricture.Some writers have seen it,they say,associated with a simple mucous urethral discharge in men of gouty habit,married and free from the suspicion of specific infection.Such discharge has been attributed to gouty irritation,to dietetic and venereal excesses,and to the contact of non-specific vaginal secretions,and such origin,in the opinion of the reporters of the cases,is well established. A gouty taint is said to be often present in this disease.

Great variation is exhibited as to the stage of the urethral disease at which the articular complication makes its appearance.It frequently sets in from the sixth to the sixteenth day of the discharge;it is common enough between the third and sixth or twelfth weeks,and may be delayed as late as the twelfth month.There is no constant relation between the severity of the urethral inflammation and the frequency with which,or the time at which,the articular symptoms arise;and these,once established,appear to be largely independent of the state of the urethra.On the adevent of the articular affection the discharge usually continues as it was,although it often abates somewhat.Fresh attacks of gonorrhoea,even when mild,oftendevelop new invasions of the joint disease, as though there were an idiosyncrasy in existence.

M O R B I D A N A T O M Y.

The usual appearances of simple synovitis are presented by the affected joint,and the capsule,synovial membrane,and ligaments become thickened and inflamed. There is often effusion into the joints,but although the fluid may appear turbid with leucocytes and fibrin, it is rarely purulent.One may make exploratory aspirations in protracted cases,thinking pus is present,but will seldom find it.It is more apt to appear in the wrist-joint than any other,for some unknown reason.The gonococcus,first found in the fluid by Petrone and Kammer,is present in only a limited number of instances, and special toxins have not yet been isolated;nevertheless,there seems good reason to believe that it is the sole cause of the arthritis,either directly or through absorption of its toxins from the local inflammation in the urethra.When pus is found in the fluid,it is due to pyogenic organisms or toxins which have made their way in with the gonococci.Staphylococci,streptococci,and pneumococci have all been discovered within the joint cavities.In most cases the inflammation is not confined to the fibrous structures within the joint,but becomes periarticular,and extends for some distance along the tendon sheaths,or sometimes the periosteum,with considerable infiltration and oedema.In this manner the gonorrhoeal joint acquired a somewhat different appearance from the acute rheumatic joint,the swelling of the former being more fusiform,less circumscribed,extending

for some distance beyond the joint, and gradually tapering off. In this way the entire dorsum of the hand and the fingers to their tips become uniformly swollen, so that the natural curve of the wrist is obliterated, and the ankle is affected in the same way. The tissues eventually acquire a much more boggy feel than in rheumatism, and one is often deceived by the impression of apparent fluctuation, which, however, does not usually exist except in a bursitis about the knee. The elbow-joint may be so swollen as to make the arm appear like a cylinder, the natural bony outlines being wholly obscured. In one form of gonorrhoeal arthritis, usually of very chronic type, there is much effusion, constituting a true hyrdarthrosis. Such cases are commonly localised in the knee, whereas the inflammation tends more towards oedema in the wrist and ankle. As the inflammation subsides, the joint recovers less completely than from ordinary rheumatism, and fibrous adhesions and thickenings remain for long, and may give rise to much impairment of motion or even pseudo-ankylosis.

S Y M P T O M S.

Gonorrhoeal arthritis may be seen in connection with any articulation; it most often invaded the larger joints at first, more especially the knee; the ankle next in order of frequency, and then succeeds the shoulder, closely followed by the smaller joints of the hands and feet, which are very seldom affected primarily and antecedently to the larger joints. The tempero-maxillary, the sacro-iliac, the sterno-clavicular, the intervertebral, do not escape gonorrhoeal rheumatism more than they do rheumatoid or pyaemic arthritis. The disease most frequently invaded several joints simultaneously or successively, but, soon declining in many of them, it finally becomes localised in a few or rarely in a single articulation. It is monoarticular from the first in about twenty per cent. of cases, especially in the knees.

Finger (Quoted by Marshall,- Syphilology and Venereal Disease, p. 469, 1906) has studied three hundred and seventy-six cases, and states that the relative frequency of the articulations involved was as follows:

Joint.	Number of Cases.
Knee	136
Ankle	59
Wrist	43
Digits	35
Elbow	25
Shoulder	24
Hip	18
Tempero-maxillary	14
Metatarsal	7
Sacro-iliac	4
Sterno-clavicular	4
Chondro-costal	2
Intervertebral	2
Crico-arytenoid	2
	376

Fournier's tabulation(New Dict.of Medical & Surgical Practice,T.v,p.230)of relative articular frequency of affection is as follows:

Joint.	Number of Cases.
Knee	73
Ankle	32
Fingers and Toes	23
Hip	16
Wrist	14
Shoulder	12
Elbow	11
Tempero-maxillary,etc.,	6
	197

Gonorrhoeal arthropathies may be clinically divided into several types as follows:

I. Arthralgia.- In this form of gonorrhoeal rheumatism there are pains of greater or less severity,sometimes increased by movement,but unaccompanied by redness or swelling;they affect one or frequently several joints; they wander from joint to joint,are liable to exacerbations,and sometimes resist treatment.This form occurs either in a chronic state in the course of an old gonorrhoea,and without other rheumatic symptoms,or as an acute affection along with other rheumatic symptoms as in the second form.Myositis or tenosynovitis may accompany it.

II. Rheumatic Type.- In this the symptoms are almost identical with those of subacute articular rheumatism or the more active forms of polyarticular rheumatoid arthritis.It was first described by Fournier (loc.cit.),and in it several joints are usually implicated,perhaps suddenly,either quite spontaneously or after chill,exertion,or strain,or rheumatic-like pains having been felt for two or three days in the soles, ankles,or loins,the painful joints become moderately swollen,tender,and hot;pyrexia supervenes with its early chilliness,malaise,and anorexia;the temperature is not high;the profuse sweating and the very acid,high-coloured urine of acute articular rheumatism are not observed,or but transiently or to a very slight degree. In a few days the moderate febrile disturbance subsides,but the local inflammation persists,and extends to other joints,without promptly leaving those first invaded;while lingering in all it often fixes itself in one or more joints,and is apt to produce a copious and rebellious intra-articular effusion.Still,it very rarely involves as many joints as primary acute rheumatism. The periarticular tissues are usually more involved than in subacute or even primary articular rheumatism. Hence the considerable swelling from oedema of the back of the hand or foot,around the knee,behind the elbow, and the copious effusion into the adjoining bursae and tendinous sheaths,and in the case more especially of the small joints of the fingers and toes the fusiform enlargement and deformities resulting from periostitis of the articular extremities.The pain,deformity,pseudo-ankylosis,etc.,produced by these periarticular processes are very persistent and rebellious,and,although they do usually disappear at last,occasionally the inflammatory irritation extends to the cartilaginous and osseous structures,and rheumatoid arthritis with its permanent deformities results.It is perhaps chiefly in

this polyarticular deforming variety of gonorrhoeal rheumatism that cerebral, spinal, cardiac, pleural, and ocular complications most frequently occur. When attacking the great toe it may simulate gout; and it differs from rheumatoid arthritis in affecting one or two or three digits only. Fournier describes instances coinciding with other symptoms of gonorrhoeal arthritis, and occurring with successive attacks of the specific urethral inflammation, and adds that the "successive repetition of the same phenomena under the same conditions shows their pathogenic connection."

III. Arthritis.- In this third variety, or acute gonorrhoeal arthritis, which is the most common and constitutes the so-called gonorrhoeal rheumatism, after two or three days of pain wandering from joint to joint, a single articulation suddenly, and frequently about the middle of the night, becomes the seat of atrocious and abiding pain, followed in a few hours by very considerable swelling of the articulation, not due chiefly to the articular effusion, but to periarticular oedema and enlargement of the bones. The pain and tenderness are most severe at the line of junction of the articular surface; the swelling begins at that point, and extends widely, especially over the dorsal aspects of the wrists and elbows, the joints most liable to this form, although any articulation may suffer. The joint is also hot, it may be pale, but is usually more or less red, and occasionally presents the appearances of severe phlegmonous inflammation, and excites a sensation of pseudo-fluctuation. The affection may resolve, or fibrous ankylosis may ensue, or very rarely suppurative destruction of the articulation may occur, although such issue has been denied by Fournier, Rollet, and Voelker. It is remarkable that, like the other forms of gonorrhoeal rheumatism, the acute inflammatory form is not accompanied by a general febrile disturbance at all proportionate to the local disease. The affection usually makes it appearance during the course of a posterior urethritis, but may occur in the first week of the gonorrhoeal attack, myositis and tenosynovitis being common accompaniments of the condition. A characteristic sign of the affection is pain in the heel, which is due to involvement of the osteofibrous attachments to the calcaneum and of the internal plantar nerve. The occurrence of suppuration may be referred to secondary infection with pyogenic micro-organisms.

IV. Hydrarthrosis.- Although occasionally accompanying the polyarticular variety, chronic hydrarthrosis is frequently observed independently, and is then often monoarticular, and affects especially the knee; however, both knees are sometimes involved. The ankle- and elbow-joints suffer much less commonly than the knee. The effusion into the articulation takes place insidiously, although rapidly producing considerable enlargement of and fluctuation in the joint, without local heat, redness, or tenderness, and often with but little or no pain or pyrexia. It is not as often associated with inflammation of the tendinous sheaths and bursae or of the eye as the polyarticular form, but it is apt to be very slow in resolving, and may last for two or three months, a year, or several years, and in scrofulous patients may degenerate into white swelling. The formation of pus in the joint is very rare. It occurred twice in ninety-six

cases tabulated by Nolen;hydrarthrosis obtained twelve times;chronic rheumatism or arthritis deformans five times;and serous synovitis sixty-four times and tumour albus once.A weak joint often remains with the patient for life.

V. Mixed Type.- In this fifth variety of gonorrhoeal rheumatism,as in the case of other varieties of so-called secondary rheumatism,the process predominantly the tendons and tendinous sheaths,the bursae and periosteum,sometimes without,but far more frequently in association with,affection of the joints.Pain,sometimes severe and increased by movement and pressure and aggravated at night,with local swelling and tenderness,are the symptoms.In their fixity and persistence,their tendency to relapse,and their chronic course these periarticular affections resemble gonorrhoeal inflammation of the joints.Gonorrhoeal bursitis is often severe enough to resemble phlegmon,but it does no end in suppuration;it is most common in the bursae covering the patella,the olecranon,and especially in that under the tendo Achilles and the deep one covering the inferior tuberosity of the os calcis;but any of the bursae may suffer from gonorrhoeal rheumatism.The periosteum in the vicinity of the affected articulation and over the most prominent parts of the bones is sometimes the seat of small circumscribed firm nodes which are painful and tender,and may either resolve rapidly or very slowly subside.Fournier(loc.cit.)first drew attention to this condition.

COMPLICATIONS AND SEQUELAE.

There are numerous complicating or consecutive condition which may be observed in connection with the disease under consideration.It is mainly in the third form of gonorrhoeal rheumatism,or independently,that the various muscles and nerves may be the seat of mylagia and neuralgia. The sciatic nerve is specially liable.

In the same form are often encountered those ocular affections observed not infrequently in rheumatoid arthritis and very rarely in acute articular rheumatism, namely,conjunctivitis and iritis.Aqua capsulitis is more common than the others,according to Fournier(loc. cit.).The ocular affection may precede,accompany,or alternate with the articular,and,not being due to direct introduction of the urethral contagium into the eye,are regarded as manifestations or localisations of gonorrhoeal rheumatism.

The forms of erythema sometimes present in primary acute articular rheumatism are now and then encountered in gonorrhoeal arthritis.

Authorities are not agreed as to whether inflammations of the heart,lungs,and serous membranes occur as manifestations or localisations of true gonorrhoeal rheumatism.Even those who,like Besnier(loc.cit.)

contend for the rheumatic nature of gonorrhoeal rheumatism admit that they are quite exceptional in that affection.Endocarditis is probably more frequent than pericarditis,and the aortic more liable than the other valves to suffer.Gonorrhoeal endocarditis has been observed without the articular affection,although it is especially when several joints are involved and the pyrexia is well marked in gonorrhoeal rheumatism that the above visceral complications occur.While admitting that Morel(Rev.des Sci.Méd.),Marty(Arch.Gèn.de Méd.,Dec., 1876),Pfuhl(Deut.Zeit.f.Pract.Med.,1878,No.50),and others have reported what appear to have been authentic cases of gonorrhoeal endocarditis,it would seem reasonable to suppose that it must be almost impossible at times to distinguish a polyarticular acute gonorrhoeal from ordinary acute articular rheumatism,and that in other instances the possibility of pyaemia developing in gonorrhoea,and producing both the articular and visceral lesions,or the latter only,cannot be denied.

The same statements are applicable to the cerebral and spinal affections which various observers have reported in connection with this disease.

D I A G N O S I S.

GENERAL CONSIDERATIONS.

It may happen that what appears to be undoubtedly ordinary gonorrhoeal rheumatism is really really pyaemic arthritis,owing to the coexistence of urethral discharge.The intermediate link in the causation may be suppuration in the prostate or its veins or in the testicle or penis or in its dorsal vein,or the urethral pus may undergo changes and become septic and be absorbed.In other instances it is highly probable that true primary acute articular rheumatism sometimes occurs coincidently with gonorrhoea.If in addition to the presence of recent existenceof gonorrhoea the case present several of the following features,gonorrhoeal rheumatism may be said to exist:,oderate or mild pyrexia and articular pain;the number of joints attacked being few, with a tendency to concentration in one,either from the first or secondarily;no migration from one joint to another;no delitescence,but marked chronicity and indolence,with a tendency to hydrarthrosis,and to implication of the synovial sheaths and bursae;an absence of cardiac complications;the frequent and often early coincidence of special ocular troubles.In all cases of gonorrhoea in men and others,the urine should be examined for filaments,the discovery of which will suggest the nature of the disease.

DIFFERENTIAL DIAGNOSIS.

SUBACUTE RHEUMATISM.

In this affection several articulations are involved,and the affection generally migrates from one joint to another,the wrists and the ankles being more often implicated than the knees;whereas gonorrhoeal

rheumatism rarely involves more than two or three joints, is not infrequently located in one joint, usually the knee, and has less tendency to wander from articulation to articulation. In subacute rheumatism there is great pain in the absence of movement; whereas in gonorrhoeal rheumatism the pain, in the absence of suppuration, is slight when the joint is kept at rest. There is usually well-marked fever, as well as profuse and sour perspiration in subacute rheumatism; whereas in gonorrhoeal arthritis there is little or no elevation of the temperature. In subacute rheumatism the urine is concentrated and possessed of febrile characteristics; in gonorrhoeal rheumatism the urine very often contains filaments from posterior urethritis, though otherwise normal. Gonococcal origination obtains in gonorrhoeal arthritis; in rheumatism of subacute type it does not.

SYPHILITIC RHEUMATISM.

Here there is less effusion, fewer joints are affected, there is no pain, and other signs of secondary syphilis are observed. Gummatous arthritis presents characteristics which eliminate it entirely from the diagnosis of gonorrhoeal arthritis. Still, a patient may have rheumatism and syphilis at the same time; the therapeutic test will then prove of service.

GOUT.

Gouty arthritis of the knee, which is rarely seen by itself, may simulate gonorrhoeal synovitis in its early stages, but the history and the course of the two affections differ entirely. Furthermore, in gout tophi will sooner or later appear, and constitutional and visceral manifestations will be of a distinctive character.

PYAEMIA.

Pyaemic synovitis may present a resemblance to the gonorrhoeal condition; but the history, the absence of urethritis, and the constitutional symptoms of chills, fever, sweating, etc., will effect the differentiation.

P R O G N O S I S.

In cases of gonorrhoeal rheumatism the recovery is very tedious, but is often complete, and one may see not a few hopeless-looking articulations eventually become normal. Provided it is once cured, the affection may return without a fresh gonorrhoeal infection, and Osler (loc.cit.) reports a case which with relapses lasted for ten years, but usually recovery is not followed by relapse without re-infection. Broadhurst reported a case of ankylosis occurring after three attacks of gonorrhoea, and fibrous thickenings may remain for a long time, producing stiffness and immobility, or adhesions may require a surgical operation for their relief. Life is not endangered, except in the very rare cases in which cardiac or cerebral complications exist.

TREATMENT.

MEDICINAL TREATMENT.

The gonorrhoeal inflammation of the urethra should be treated in the same way as it ought in the absence of arthritis. Rest, moderate diet, and even the salicylate of soda, favour its removal; but the frequent employment of mild astringent injections, balsamic preparations, and the usual medicaments required for the parent disease should not be omitted.

Salicylate of soda, given freely, is sometimes signally useful, more especially when several joints are acutely inflamed. In the more chronic stages, when much articular effusion exists, a prolonged course of iodide of potassium us occasionally beneficial. Iron and quinine will be demanded for the relief of the general debility, anaemia, and impaired nutrition; and the same may be said of cod-liver oil, extract of malt, etc. Various baths may do good as in chronic rheumatism. Iodide of potassium should not be given in the acute stages lest it make the urethritis worse. Morphia may be required for the first few days to relieve the pain. Wilson (Jacobi's Festschrift, 1900) says that he has had excellent result from the employment of massive doses of the syrup of the iodide of iron. The bowels should be kept open in the usual way, and the diet should be of the most nutritious kind.

LOCAL TREATMENT.

The treatment of gonorrhoeal rheumatism is mainly local, and its principles and details are much the same as those indicated for rheumatoid arthritis which it so closely resembles, and which will be presently described. Very satisfactory results may be derived from the continuous application of cold in the form of ice-bags, or ice poultices, made by mixing pounded ice with linseed meal, by which means the ice does not melt too rapidly and the cold can be uniformly and conveniently applied. For the first few hours the pain may be increased, but relief is almost sure to follow, and the treatment should be persevered in for weeks together, if need be. Guaiacol and glycerine in equal parts, or a forty-per-cent. ointment of ichthyol with lanolin, may be smeared over the inflamed area. Dezanneau (La Scalpel, Oct. 4, 1896) strongly recommends the topical application of turpentine with an equal parts of a watery emulsion of green soap.

Absolute immobility of the joint must be secured by splints, or in the case of the knee, sandbags should be used, while the weight of the bedclothes is kept off with a cradle. As the very acute symptoms subside, it may be well to encase the joint in plaster of Paris for a week or ten days, but not for long, lest adhesions form which are difficult to eradicate. This formation can usually be prevented when pain is gone and the swelling has in great part subsided by abandoning cold and massaging the joint after soaking it for ten minutes

in water as hot as can be borne.This should be done two or three times a day.Injections of corrosive sublimate have been recommended. Hydrarthrosis may be diminished by the use of Paquelin's thermo-cautery or blisters at intervals.The patient must,of course,be kept in bed from the beginning to the ending of the attack.

The ocular and other complications may be treated on general principles.Conjunctivitis requires protection from light,cold applications,and the instillation of a saturated solution of boric acid.Atropine should be used if iritis be present.

RHEUMATOID ARTHRITIS.

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SYNONYMS.

Arthritis Deformans; Rheumatic Gout; Nodular Gout; Nodular Rheumatism; Osteo-arthritis; Chronic Rheumatic Arthritis; Progressive Chronic Articular Rheumatism; General and Partial Chronic Osteo-arthritis; Arthritis,Rheumatisma Superveniens(Musgrave); Arthritis Pauperum; Arthritis Sicca; Nodosity of the Joints (Haygarth); Usure des Caetilages Articulaires(Cruveilhier); Arthrité Chronique(Lute); Rheumatisme Noneux; Goutte Asthénique Primitive; Polypanarthritis(Heuter); Arthritis Spuria; Arthritis Nodosa; Arthritis Rheumatoides; Arthritis Anthroxerosis; Malum Senile Articulorum.

DEFINITION.

Rheumatoid arthritis is a very chronic affection of the joints,characterised by the production of great deformity,exhibiting slowly progressive changes in the synovial membranes and articular cartilages,with extensive osseous periarticular growths. A somewhat acute course is pursued in quite exaceptional instances.

HISTORY.

Though the knowledge of rheumatoid arthritis has had comparatively recent beginnings,its history can be traced back,in a fragmentary manner,to a very remote period of antiquity.Bones bearing the unmistakable stamp of this disease were unearthed by Petrie(Brit. Med.Jour.,1891,ii,p.658)in the course of his explorations in Egypt,and some of the bones so marked,which he found,date back in all probability to the very dawn of Egyptian history;while others have been discovered by Eve(ibid.,1890,i,p.433)which were probably those of persons who lived not earlier than Ptolomaic times.

Similar evidences have been discovered by della Chiagi, in bones found amongst the ruins of Pompeii, by Moore (Trans.Path.Soc.,1883,xxxiv,p.226) in the bones of a human being discovered in a Roman sarcophagus in London; and by Virchow in those fromthe of an ancient Pomeranian burial-ground.

The older writers on medicine were well acquainted with the disease, for example, Musgrave(De Arthritide Symptomatica,1703,p.24), de Sauvages(Nosologica Methodica, 1768,Class vii,Ordo 1), and Sydenham(Opera,1633,Sect.vi, Cap.V), as well as others. The latter's account is particularly noteworthy; when speaking of rheumatic fever he describes as an occasional legacy of that disease a condition of the joints which may persist for years or even throughout the life of the patient, characterised by distortion and deformity with nodular outgrowths of the joints of the fingers and other articulations, which tend to cripple the patient more or less completely, although there may be little or not any affection of his general health. He described the condition in 1766-69, and appears to have clearly distinguished it from gout. In 1800 Landré-Beauvais(Is there a New Kind of Gout? Thèse de Paris,1800,Aug.3) read a paper before the Faculty of Medicine of Paris, on a form of articular disease to which he gave the name of goutte asthénique primitive; and it has been claimed by Charcot for this contribution that it embodies the earliest description of rheumatoid arthritis as a distinct and morbid entity. This claim is to a great extent justified by the fact that Beauvais clearly described the changes which this form of arthritis produces in the structures of the joints which it attacks, such as the destruction of the articular ends of the bones, apart from any deposit of tophaceous material. Yet, when we consider the clinical descriptions of cases contained in his paper, it is impossible to suppose that more than a comparatively small proportion of the cases included were actually examples of rheumatoid arthritis; some of them were undoubtedly examples of true gout, and other perhaps of diseases of doubtful pathology. His ideas as to the symptoms of the disease were of a decidedly vague character, though he certainly deserves the credit of having been the first to recognise its lesions.

In 1804 Haygarth(On Nodosities of the Joints, Clinical Histories of Diseases,1805) pointed out some of the more striking clinical features of this disease, and distinguished it from both gout and chronic rheumatism under the title of nodosities of the joints. He studied some thirty-four cases at Bath, and had his attention first drawn to the subject by a case which came under his care which convinced him that there is one painful and troublesome articular disease, of a peculiar nature, and clearly distinguishable fromall others by symptoms manifestly different from gout, as well as from both acute and chronic articular rheumatism. He noted the peculiar liability of the female sex to the disease, especially at or about the menopause; the special tendency of the joints of the fingers to be attacked; the characteristic enlargement of the articular ends of the bones, and the essentially chronic nature of this affection, which tends to implicate one articulation after another, until the patient may be crippled with it; and

which as it spreads does not leave the articulation relieved from its machinations.He tells us that he had written a paper upon the subject twenty-six years previously,although it was not published;and to him belongs the merit of having so enumerated its characteristics as to give it a place in nosology.His observations apparently fell on barren ground,and for many years after his literary contribution the literature is almost blank so far as references to the disease are concerned.Even Scudamore(On Rheumatism,1827,p.487),writing twenty-two years later,although he refers to the observations of Haygarth,says that he has seldom met with the condition described by that author except as a consequence of antecedent attacks of gout or rheumatism.Brodie(Diseases of the Joints,1836,p.27),on the other hand,maintained that rheumatoid arthritis was essentially different from both of those ailments.

Later the researches initiated by Beauvais were taken up by observers both at home and aborad,and vigorously pursued.Incidential allusions were made to the affection in 1813 by Chomel,in 1818 by Brodie(loc.cit.), and by Lobstein(Anat.Path.,1833,ii,p.348)and Ashton Key(Med.-Chir.Trans.,1833,xviii,p.141)in 1835;the affection was also mentioned by Robert Smith(Dubl.Jour.Med. Sci.,1835,vi,p.205)two years later,by Canton(Lond.Med. Gaz.,1848,N.S.,vi,p.410)and Deville(Bull.de la Soc.Anat., 1848,xxii,p.272;xxiii,p.141)in 1848,and by Broca(ibid., 1850,xxv,p.435)in 1850;Cruveilhier(Anat.Path.,1856,Liv., xix,p.13) in 1856 pointed out some of the most striking characters of the morbid anatomy of the disease.But it is to Adams(On Rheumatic Gout,1857;Ed.2,1873)that we are indebted for the most complete account of the amatomy and of many of the clinical features of the malady:first in a paper read before the British Association in 1836, next in his article on "The Abnormal Conditions of the Elbow,Hand,Hip.etc.(Todd's Cyclop.of Anat.& Phys.,1836-1839),and finally in his interesting contribution parenthetically quoted above.Both to him and Robert Smith we owe the recognition of the fact that the disease of the hip-joint encountered in elderly men,which so frequently has its origin in an injury,slight or sebere,is in its essential nature identical with the disease of many joints,both large and small,to which the female sex are particularly predisposed.

It may here be specially noted that both Landré Beauvais and Haygarth described more particularly that form of the disease which,beginning in the small joints of the extremities,tends to extend to the larger joints in a centripetal way,and to involve many of them - peculiarities which have given rise to the synonyms progressive polyarticular chronic rheumatism,peripheral arthritis deformans,and which is the form of the disease usually described by physicians as rheumatic gout,rheumatoid arthritis,nodular rheumatism,and by the other names just mentioned.On the other hand,Key,Colles,Adams, in his earlier paper,and R.W.Smith described the disease as it affects the larger joints,hip,shoulder,or knees, to one or two only of which it may be confined;and as this variety is frequently observed in elderly persons, and in them often involves the hip,it is often spoken of as senile arthritis,mono-articular arthritis deformans, partial chronic rheumatism,and has been described by

surgeons rather than by physicians.However,even when beginning in the hipor shoulder,the disease is apt to involve several of the intervertebral articulations,and not infrequently to extend to other joints than the one first affected,and even to the peripheral joints.Its progressive and general nature is thus evidenced,whether it invade from the beginning a single large joint or several symmetrical small articulations.

Charcot(Thèse de Paris,1853)insisted that Heberden's nodi digitorum contributes a special form of the disease under consideration,and proposed to call it Heberden's rheumatism or nodosities.According to him (Lectures on Senile Diseases,1881,Syd.Soc.Ed.,p.137)and Trastour(Thèse de Paris,1853),rheumatoid arthritis is but a variety of chronic rheumatism,which theory was widely entertained by contemporaries abroad until a comparatively recent period and is still believed in by several writers.In the course of Charcot's narrative we find graphically described and classified into definite types the deformities of the hands which result from changes in the joints,and from the spasm of the muscles which have to do with the performance of the functions of the digits.In 1852 and 1859 Fuller(On Rheumatism,Rheumatic Gout,and Sciatica,Ed.3,1860)and A. B. Garrod(Gout and Rheumatic Gout,1876)contended that arthritis deformans is a distinct disease differing from both rheumatism and gout in its pathology,laying particular emphasis on the fact that the visceral manifestations of true rheumatism are not met with in rheumatoid arthritis,and that in the latter there is no excess of urate in the blood as obtains in gout.

Those who accepted the theory that the affection has nothing to do with either gout or rheumatism insisted that the synonyms suggestive of such a relationship should be abandoned.It is an independent disease of unknown origin,although its coexistence with either chronic rheumatism or gout is possible.Of the latter, William Ewart(Gout and Goutiness,p.161)writes:"The coexistence with gouty deposits of changes resembling osteo-arthritis led Fuller and Hutchinson to believe in a blending of the rheumatoid with the gouty element,at least in some cases.This is the view also taken by the writer(Ewart).Mixed forms may arise in which the osteo-arthritic changes are conspicuous,and uratic incrustations may also have occurred.This in no way invalidates the statement as to the independent nature of the two diseases;indeed,their dualism always asserts itself either clinically or in their anatomical appearances!" We have seen that in 1805 Haygarth believed that the disease was "clearly distinguishable from all others by symptoms manifestly different from the Gout and from acute and chronick Rheumatism".E Wynne(Lancet,1889,i, p.933)has demonstrated that even in those severer cases of gout,in which there is cartilage erosion and lipping of the periphery of the bones,there are always pathological features which are absolutely distinct from those of rheumatoid arthritis.It is quite possible to believe that in some cases repeated attacks of acute rheumatism or gout may,like other injuries,by the insult offered to the joints,pave the way for the localisation in them of subsequent trophic changes peculiar to arthritis deformans,and this in nowise calls for the belief

in a common origin of any of these affections.

The next step in the history of the disease was the suggestion of a nervous origin for it;this was first done by Mitchell,and Ord(Trans.Clin.Soc.,1879,xiii;Brit. Med.Jour.,1884,ii,p.263),Sir Dyce Duckworth(Liverpool Med.-Chir.Jour.,luly,1891,p.245),Weber(Jour.Nerv.& Ment. Dis.,1884,N.S.,Vol.ix,p.72),Garrod(loc.cit.),and others have adduced many facts in support of a neuropathic theory of origination,the most striking of which are as follows:bilateral symmetry in the distribution of the lesions;associated trophic changes,notably in the skin and nails;disproportionate muscular atrophy,Ord dwelling upon the resemblance of the latter to progressive muscular atrophy,and suggesting that the primary cause may reside in the trophic centres of the spinal cord,or that it may be due to peripheral irritation from trauma,uterine or urethral lesions,etc.;frequent persisting mental disturbance,such as shock,worry,grief;and the fact that certain cold lesions are productuve of arthrophathies or dystrophies,such as are observed in locomotor araxia with the symptom known as Charcot's disease,syringomyelia.In two autopsies made by Falli(Il Policlinico,Dec., 1894)upon typical cases of arthritis deformans there was atrophy in the anterior horns of the cord,through which trophic impulses pass outward.In one of these cases degenerative changes were also present.Pitres and Vaillard(Rev.de Med.,1887,Vol.vii,p.456)have described concurrent lesions of neuritis in several instances of rheumatoid arthritis,and the centripetal progression of the disease from the periphery in its multiple type accords with the theory of nervous origin,which is now widely entertained.

Dor and others claim to have proved that the affection is due to a specific bacillus;but their researches as yet lack confirmation.

P A T H O L O G Y.

GENERAL CONSIDERATIONS.

The pathogenesis of arthritis deformans is the subject of differences of opinion very like those existing in regard to acute articular rheumatism.Numerous observers have been in favour of its relationship diathetically to rheumatism,they affirming that the doctrine of an arthritic diathesis appears to be specially applicable to it,with less difficulty than to acute rheumatism,and that the probability of a specific germ being its true cause is very remote.They say that what seems to be necessary to the foregoing is,that the causes shall be be more persisting and oft-recurring, so as to maintain a prolonged local irritation of the articular tissues,or that the neuro-arthritic diathesis shall be highly developed;under these conditions the prolonged or oft-repeated application of cold and damp to the peripheral nerves,severe or oft-repeated slight injuries to joints,urethral or ovarian irritation, chronic gout or rheumatism,or even,exceptionally,an

attack of the acute forms of these diseases,may originate rheumatoid arthritis;and all wearing influences, such as anaemia,excessive menstruation,prolonged lactation,innutrition,failing health,mental anxiety,or shock,etc.,act as adjuvants in the development,aggravation,and maintenance of the joint affection.

THEORETICAL CONSIDERATIONS.

Several theories have from time to time been advanced to account for the origination of rheumatoid arthritis. They are as follows:

Dystrophic Theory.- This hypothesis refers the arthropathies of rheumatoid arthritis to reflex or other dystrophic nervous lesions;and the idea was suggested by the similarity of the lesions to those found in certain forms of arthritis occurring during the course of organic affections of the nervous system,such as tabes dorsalis and syringomyelia,which are generally believed to be dystrophic in nature.The advocates of this theory lay stress upon their affirmation that the striking symmetry observed in the distribution of the lesions in the multiple form of the disease is also most readily explained on the theory of a nervous origin, the degree of symmetry attained to being much greater than in other forms of joint affection.But unless we are prepared to assign all symmetrical diseases to such influences,or at least to suppose that the distribution of the lesions is controlled by the nervous system,we must be careful not to press this argument unduly.It is as well to bear in mind that the subcutaneous fibrous nodules of young rheumatic subjects,and various forms of skin disease which are not,like shingles,obviously closely dependent upon nervous lesions,are apt to exhibit a curious symmetry of distribution,which may sometimes be comparable to that shown in rheumatoid arthritis;and that in tabes dorsalis at any rate the joints are not particularly apt to be symmetrically invaded,although in some other types of organic nervous disease there may be involvement of corresponding joints on the two sidesof the body.The nervous theory is also quite compatible with the early invasion of the peripheral articulations,and the tendency of the affection to progress centripetally;in addition to which,the etiology of the malady would seem to indicate that its causes are such as might reasonably be expected to give rise to a nervous affection rather than to some other form of constitutional disease;and,moreover,when the changes in structures other than the articulations which are associated with rheumatoid arthritis are considered,it is found that the visceral lesions which often play so conspicuous a part in diseases of the blood are absent; whereas a dystrophic character is assumed by the changes in the muscles,skin,and other tissues.The muscular atrophy is probably of this character,though these structural alterations are certainly to some extent secondary to the articular lesions and are seen in both chronic and acute forms of joint disease.Numerous writers,however,are firm in their insistence that there is in rheumatoid arthritis something more than mere arthritic atrophy,and that to some extent at any rate the muscular wasting is more merely secondary to,but advances pari passu with the joint disease,and may even be encountered in parts in which the articulations do not

as yet manifest any evidence of morbid alterations. In certain instances dystrophy of the general integument, disturbances of skin pigmentation, local sweating, numbness and tingling of the hands and feet may be observed; and these ailments are recognised accompaniments of nervous disease. On the other hand, objectors to the nervous theory urge that no anatomical evidence of disease in the spinal cord, to which the articular dystrophy can be referred has ever been found. Pitres and Vaillard state that they have come across changes in the peripheral nerves, and think that these may play some part in the causation of the cutaneous and muscular changes at least. They found that in a case in which muscular atrophy was very marked the muscular nerves were profoundly affected, whereas in a second case, in which the atrophy was slight, they exhibited no evidences of neuritis. The nerves supplying the affected articulation were but little affected, and our authors hold that to this cause the articular lesions themselves cannot be referred. According to Ord (loc.cit.) a refle theory best explains the symptoms of rheumatoid arthritis; he thinks that the uterus is very often the organ from which the centripetal influences which are reflected along the same paths as those which in primary diseases of the spinal cord lead to articular disease. In support of his contention he adduces evidence of the important part played by uterine disorders in the causation of theumatoid arthritis, and suggests that predisposing conditions, such as debility and anaemia, may assist this reflex process by increasing the morbid reflex excitability of the spinal cord occasioned by the affection of the uterus concerned. This nervous theory, though convenient, is not yet definitely established owing to the lack of pathological proof.

Microbic Theory.- This has a small, but increasing number of adherents. A definite organism is said to have been found by Dor, who claims to have reproduced the disease by injecting cultures into the blood of a rabbit, and considers the germ an attenuated culture of the staphylococcus pyogenes aureus. Dungern and Schneider isolated after death, from the mucus of the gall-bladder, and from the exudates in the joints, small diplococci that did not resemble the organisms previously described by Blaxall and Schüller. Lesions similar to those observed in the patient resulted from the injection of cultures into the knee-joint of rabbits. These researches require further confirmation and investigation.

Combined Theory.- This theory originated with Hutchinson (Med. Times & Gaz., 1881, i, p.1; Trans. Path. Soc., 1880-81, xxxii, p.193; The Pedigree of Disease, 1884, p.126; Trans. Internat. Med. Congr., 1881, ii, p.92) who believed in the blending of the rheumatic with the gouty element, in some cases at least. This idea has for its basis the existence of the arthritis diathesis through the instrumentality of which the rheumatic and gouty affections of the joints are produced. It is certainly strange to observe how rheumatism and gout are sometimes associated in a family, and it is difficult to reconcile such instances with the obvious and conspicuous differences which they otherwise present, and with the evidence which points to the possession by each disease of an entirely different pathology, and to their inclusion in quite

different groups of affections.For example,the apparent liability of the daughters of gouty parents to rheumatoid arthritis lends support to this theory;but,on the other hand,all that can be said against the rheumatic theory can be urged against this,and we have the fact observed by Garrod(loc.cit.)that no excess of uric acid is to be detected in the blood of patients suffering from rheumatoid arthritis,and the point on which Duckworth(loc.cit.)lays stress,that in countries in which true gout is next thing to unknown rheumatoid arthritis is practically unknown.Articular affections in gout are characterised by the deposition of urate of soda,but such deposits are not encountered in the joints of suffers with rheumatois arthritis unless there has been an antecedent attack of true gout;to deny that uratic deposits and the presence of uric acid in the blood are characteristic and essentials of gout is to deny the existence of that disease altogether.The thoery is therefore absolutely untenable.

Rheumatic Theory.- One of the oldest ideas regarding the pathology of rheumatoid arthritis ascribed the latter to rheumatism,which it may resemble.But in most cases arthritis deformans has its distinctive characteristics from the first,being developed de novo without any antecedent rheumatic attack;besides acute rheumatism is not the only articular affection of which rheumatoid arthritis may constitute an outcome.The rheumatic theorists point to the part played by cold and damp in the causation of the disease,and the association with articular lesions of visceral and other affections similar to those encountered in connection with true rheumatism. The general idea nowadays seems to be that the influence of cold and damp in the production of rheumatoid arthritis has been unduly emphasised;and that the endeavours to prove the occurrence in association with this disease of the abarticular lesions of rheumatism have resulted rather in the demonstration that rheumatoid arthritis is not attended with any visceral lesions other than such as may be looked upon as intercurrent affections.But,urge the advocates of the theory,what about the subcutaneous rheumatic nodules?Perhaps an exception should be made in favour of these,but our knowledge of them and of the nature of the articular lesions with which they are associated is not yet sufficiently complete to allow of any definite conclusion being drawn from their occurrence.It must,however,be admitted that at first sight the development,in association with apparently typical cases of rheumatoid arthritis,of excrescences so strangely like those which have been regarded as pathognomonic of the true rheumatic process does render the rheumatic theory rather probable.The fact that rheumatoid arthritis is most apt to commence at a period of life when true rheumatism loses much of its tendency to attack the heart,and is apt to limit its activity to the joints alone,considerably detracts from the plausibility of the argument based on the absence of visceral lesions;but the immunity from cardiac complications is quite as conspicuous in those cases in which rheumatoid arthritis is developed in early adult life,and even in childhood,if we exclude those cases in which it crops up as a sequel of rheumatic fever.Some writers hold that rheumatoid arthritis

is a variety of chronic rheumatism;but they have to admit that the chronic articular affections which follow upon the more acute forms of that disease have more commonly a different character,that,as a rule,the destruction of cartilage and tendency to the formation of bony outgrowths is wanting,fibrous ankylosis and thickening of the joint capsules being the changes more usually seen.In such cases the superficial resemblance to rheumatoid arthritis in its early stages may be very close,but,if,as is fortunately very seldom the case in the earlier stages of the affection,the case comes to autopsy,on cutting through the greatly thickened capsule there is found to be nothing wrong with the cartilages and the articular ends of the bones.

Senility Theory.- It was long ago affirmed that rheumatoid arthritis is an affection characterised by the occurrence of senile changes in the affected articulations,which,in other words,is the articular expression of the tendency of the textures of old persons to undergo degenerative processes.In the opinion of many,this theory is by no means lacking in plausibility.

Traumatic Theory.- The sixth theory advanced is that of Lane,and is an hypothesis of the occurrence of wear-and-tear,based upon the great destruction of the cartilages and osseous surfaces,and the grooving of the latter in the lines of the articular movements. But it seems to me that it is obviously incapable of explaining the multiple form of the disease,in which we find the articulations of the hand early attacked,often in those who have never done hard work of any kind,and in which the lesions show a remarkable tendency to symmetry;and Lane himself ultimately came to the same conclusion.It may be mentioned,however,that wear and tear plays a prominent part in the production of the articular changes when the joints are once attacked by rheumatoid arthritis.

M O R B I D A N A T O M Y.

GENERAL CONSIDERATIONS.

Every component tissue of the joints exhibits a chronic inflammatory process,the appearances of which have long been known.Though no structure of the joint escapes involvement,it is doubtful which one is first attacked;probably it is the cartilaginous coverings of the articular ends of the bones which see the initiation of the disease.The affection would be better understood in this and other respects were more cases to come to autopsy,it being only seldom and usually accidentally that one has an opportunity of studying the lesions at an early stage of evolution.These lesions are not absolutely characteristic of the cases to which the name of rheumatoid arthritis is usually applied;for similar alterations have been encountered in the articular troubles of tabes dorsalis,as well as in haeophilia,and in chronic and old-established instances of gout.Even amongst the cases commonly classed together as examples of rheumatoid arthritis there exist

conspicuous clinical differences, such as separate hip-joint disease of elderly men (morbus coxae senilis), from the more acute affection of many joints which is unfortunately so common among women; sometimes, again, it seems that rheumatoid arthritis developing late in life and running a very slow course is little more than a senile articular process.

THE LESIONS IN DETAIL.

The Cartilages.

The lesions of the cartilages are amongst the most characteristic phenomena of rheumatoid arthritis. At an early stage in the course of the disease the articular cartilages lose their uniform structure, and present the appearance of velvet, due to fibrillation of the intercellular substances, associated with disappearance of the cartilage cells. The earliest thing observed is the multiplication of the cells throughout the entire thickness of the cartilage, and the formation of capsules around some of them. Within these primary capsules a number of secondary capsules are formed, which may be either independent of each other or grouped together in common envelopes. Previous observers had mistaken these capsules for true cartilage cells, but Cornil and Ranvier (Manual of Pathological Histology, Ed. 2, 1881, i, p. 463) showed that this is not their real nature by staining them with iodine, which colours the protoplasm of the cartilage cells brown, but gives to these secondary capsules little or no pigmentation. The primitive capsules which approach most nearly to the surface rupture into the joint, and those which are more deeply seated, being able only to grow toward the surface of the cartilage, rupture into each other, and in this way a series of parallel tubules is formed, and the ground substance of the cartilage is split up into filamentous structures. It does not take long, however, for the velvety cartilage to become destroyed in places by the friction of the articular surfaces upon each other, leaving the underlying bone completely bare. As might be expected, the removal of the cartilage is first brought about over the central portions of the articular surfaces, but as time goes on the exposed area becomes more and more extensive. The remarkable tendency to symmetrical invasion which is so characteristic of rheumatoid arthritis is manifested even in this, and the destroyed area in the articulation of one limb may closely resemble, both in shape and position, that in the corresponding joint. Rindfleisch holds that the destruction of the cartilage is not purely mechanical, but is greatly facilitated by the process of mucous degeneration; and it is interesting to note in this connection that the presence of a large amount of mucin was detected in the synovial fluid removed from a diseased joint by Hoppe-Seyler (Virchow's Arch., 1872, liv, p. 225). Many cases are "dry," but effusion of synovia is also common, and if present it is an early phenomenon and mat later disappear.

The Bones.

The morbid alterations of the bones in this disease consist of the formation of osteophytes, eburnation, and abrasion. The heaping up or lipping of their edges is in marked contrast to the destruction of the central portion of the articular cartilages. To some extent the edges of the cartilaginous surfaces are overlapped and covered by synovial membrane, and their synovial covering

prevents the escape of the contents of the ruptured capsules into the synovial cavity,with the result that the cellular elements collect under it and lead to the formation of outgrowths of newly formed cartilage which are known as chondrophytes.As time goes on these chondrophytes become ossified and then constitute the familiar bony outgrowths or osteophytes,which have so important a share in the production of the deformities characteristic of rheumatoid arthritis.In some part,instead of definite outgrowths,a rim of newly formed bone is developed,which gives rise to the condition recognised clinically as lipping.The ossification begins in the parts in the immediate neighbourhood of the original bone,and progresses very slowly,so that,even at a period when the process is far advanced the osteophytes still retain a covering of cartilage.As a result of the outgrowth of osteophytes the bulk of the articular ends of the bones may be very greatly increased,and the mobility of the joint may be thereby seriously impaired.The bone in the more central portions of the articular surfaces is also found to have undergone a remarkable change.The superficial layer has become extremely hard and compact,so that it resembles ivory in appearance,a resemblance which is further increased by the polishing which it has undergone in consequence of the articular surfaces rubbing against each other.On this account thechanges in question have been termed eburnation.The smoothness of the eburnated surfaces is broken by minute perforations with which it is riddled,and which correspond to the Haversian canals which have been laid open.Eburnation may be regarded as,to some extent,a conservative process, because the hardness of the ivory-like layer tends to impede the process of abrasion to which the exposed articular surfaces are liable.Various theories have been advanced to explain the process of eburnation.Ziegler (Text-Book of Pathological Anatomy,1890,p.555)attributes this change to a softening of the subjacent bone simultaneously with the changes in the cartilage which lead to fibrillation,which softening leads to the production of cavities,into which a growth of vascular medullary substance extends,and in this the formation of a layer of new bone is effected.On the other hand,Cornil and Ranvier(loc.cit.)hold that the formation of the compact new bone is due to the discharge into the adjacent medullary spaces of the cartilage capsules of the deepest layers,which as they increase in size press on the interveing bone so as to cause its absorption;they do not deny,however,that the lesion may have something to do with inflammatory changes.Not infrequently even the eburnated surfaces exhibit marked signs of the abrasion to which they are subjected,and are apt to be scored by deep grooves running parallel with each other and following the direction in which the bones rub on one another in the ordinary maovements of the articulation. Serious alterations in the mechanism of the affected joints may be produced by the combined action of the two processes of osteophytic formation and abrasion. What was originally a convex articular surface may be reduced by abrasion to a concave form,and the abnormal cavity may be rendered much deeper by osteophytic outgrowths surrounding it.In the case of the shoulder-

joint for example,what was once the glenoid cavity of the scapula may become converted into a highly polished convex boss;whereas the head of the humerus may be changed into a cavity surrounded by bulky osteophytic outgrowths,which have themselves undergone extensive abrasion adapting their form to the requirements of reversed mechanism of the articulation.Volkmann(Handbook of Surgery,Bd.2,p.555)showed that the abrasion of the bony surfaces is almost certainly aided by some alteration in the structure of the bone itself,which in its earlier stages is probably of the nature of a rarefying osteitis;he observed that mere mechanical firction does not sufficiently explain the fact that grooving may take place on portions of the bony surfaces which are not yet denuded of their cartilaginous covering;and he held that a hardening process later supervenes on this rarefying change.As a result of rheumatoid arthritis true bony ankylosis is almost unknown.Still,it has been seen in the vertebral column,where this change has been described by Bowlby(St.Bart.Hosp.Reps.,1890,xxvi,p.77);but in the joints of the extremities,with their far greater mobility,it never occurs.Neither is there any tendency to the production of fibrous adhesions,a process which is so conspicuous in some forms of articular disease, and even in the articular lesions of haemophilia,which resemble those of rheumatoid arthritis in certain respects.The morbid alterations in the shafts of the long bones are far less conspicuous than those of the articular portions;yet they may be increased in size and more dense than usual,as was pointed out first by Adams(loc. cit.)

The Synovial Membranes.

These become inflamed coincidentally with the changes in the cartilages.In some cases in which operations have been performed upon articulations thus diseased during life,brilliant injection of the synovial membrane has been observed;but the most typical changes are the conspicuous thickening of the membrane,and the hypertrophy of the synovial fringes.They constitute a very strking feature of the disease.In the hypertrophied tufts fatty changes frequently take place,or they may become converted into cartilaginous masses which remain attached to the surface of the membrane by slender pedicles.The latter are very apt to be ruptured,with the result that the pellet of cartilage becomes converted into a loose body lying free in the cavity of the joint. The bursae in the neighbourhood of the affected articulation may also contain similar cartilaginous masses; they are easily and often to be detected by palpation in the bursa over the olecranon.It seems strange that some of the older writers should have maintained that one of the characteristics of rheumatoid arthritis is the absence of fluid in the cavities of the affected articulations;one of the names assigned to the disease, namely arthrite sèche,was intended to emphasise this view.This statement is quite opposed to the facts,and effusion,often of considerable amount,is frequently present both in the cavities of the joints and in the bursae in their immediate neighbourhood,many of which are doubtless in direct communication with the articular cavities.According to Hoppe-Seyler(loc.cit.),the articular fluid from a case of arthritis deformans

contains(in parts per 1,000):mucin,28.19;albuminous substances,20.92;ethereal extract,0.93;alcoholic extract, 1.30;watery extract,0.65;acetic extract,1.53;and inorganic substances,8.79 - making the total solids 62.31 and water 937,69.Cholesterin,lecithin,and traces of fat were found in the ethereal extract by this observer.

The Muscles.

The muscles also undergo very characteristic changes in rheumatoid arthritis,a general muscular wasting being observed.The affected muscles are,on examination after death,found to be atrophied and to have a brownish tint comparable to that of dead leaves.This atrophy is of the type encountered as the result of nervous lesions,the degeneration of the various component fibres being conspicuously unequal in degree.Some of the reported cases have presented changes in the nature of a neuritis,the amount of which corresponds to the degree of muscular wasting observed.

Visceral Lesions.

Most of the visceral lesions which have been discovered after death may be ascribed to intercurrent diseases rather than to rheumatoid arthritis.Pulmonary tuberculosis andchronic interstitial nephritis are among the most common of the changes that arethus brought to view.Cardiac lesions are rare,and are probably due to atheroma or to renal disease or to previous attacks of articular rheumatism.

E T I O L O G Y.

We have seen that the causation of rheumatoid arthritis is involved in much obscurity,various theories being from time to time advanced to account for same. We shall first examine the general progressive form,or multiple arthritis deformans,which is the most common; and then inquireinto the origination of the partial form,concluding with the etiology of Heberden's nododities.

(A)GENERAL OR POLYARTICULAR OR MULTIPLE AND PROGRESSIVE FORM.

AGE.

Age is not without influence in the production of this disease,it being oftenest seen between twenty and thirty;it continues to occur frequently up to the period of the menopause,fifty,after which it develops comparatively seldom.Of Ord's(Brit.Med.Jour., 1880,156)thirty-three cases,ten were between twenty and thirty years;eleven between thirty and forty;nine between forty and fifty;and three between fifty and sixty. It occasionally begins in both sexes after sixty,and a first attack has been reported after eighty.On the other hand,children are not exempt.Seguin(Med.Rec.,1877, 797)saw three children of the same family suffering from the disease at ages from two and a half to four years.Moncorvo(On Chronic Rheumatism,etc.,Paris,1880) met with an example at two years and a half,Laborde at four,and Charcot at ten.Osler(Practice of Med.)reports four cases under twelve years.It has been observed

that in women the majority of cases begin during or after the menopause, and in other cases which begin earlier exacerbations occur at this time of life.

CLIMATE.

It has been maintained that rheumatoid arthritis is more frequently observed in cold and damp climates than in those of opposite qualities, by those who hold that cold is its most common cause. However, it is met with in India and other hot climates. Besnier says that it is almost unknown in the tropics; but in this he has now and then met with contradiction.

HEREDITY.

It would seem that hereditary influences are responsible for the production of some cases of rheumatoid arthritis, although this factor does not obtain to anything like the same extent as in gout. Sir A Garrod (loc.cit.) says that hereditary predisposition exercises but very little influence; we certainly often see the disease confined to a single member of a large family, although Seguin (loc.cit.) saw three children of tender years in one family suffering from it, their parents being free from any disease. Trastour (loc.cit.) three times saw the children of women who were afflicted with nodular rheumatism already suffering from articular rheumatism; and Charcot (loc.cit.) once saw the grandmother, the mother, and the granddaughter successively attacked. Cases have appeared in the literature in which the patient's mother at fifty-five and the maternal grandmother at sixty became subjects of crippling polyarthritis; and others in which the mother and a young sister were, like the patient himself, victims of the disease. This direct transmission appears to be rare, judging from my own experience and the few instance of it mentioned by writers. But very many authorities maintain that simple acute and chronic rheumatism and gout in the parents predispose to rheumatoid arthritis in the offspring - Charcot, Trastour, and Besnier, for example. Now, the facts given in support of this opinion are not numerous. Trastour found that out of forty-five cases of nodular rheumatism the father or mother were rheumatis in ten instances, but the form of the rheumatic affection is not stated. Charcot, Besnier, and Homolle (loc.cit.), although believers in this doctrine, do not cite an example in proof. However, in Pye-Smith's (Guy's Hosp.Reps., S.3, xix, 348) twenty-seven cases of osteo-arthritis, five stated that rheumatism had occurred in their families. Thus, two fathers had had rheumatis fever, and one was rheumatic, and two sisters of different families had had rheumatic fever. Besides, the father of a sixth and the grandmother of a seventh had had gout. The evidence in favour of the doctrine that true articular rheumatism transmits an hereditary tendency to rheumatoid arthritis does not appear to be conclusive, although it is highly thought of by those who regard the latter disease as a variety of rheumatism. Some considerations of an opposing character deserve mention. Acute articular rheumatism has very rarely passed continuously into rheumatoid arthritis, and very rarely has been followed at short interval by that disease; and in such exceptional cases the antecedent affection may have been really the acute form of rheumatoid arthritis, which closely resembles acute articular rheumatism. Trastour (Thèse de Paris, 1853, p.41), Vidal (ibid., 1855, p.9), Charcot (Clinical Lectures, p.214) and

others admit that acute rheumatism can hardly be placed amongst the antecedents of the rheumatoid affection.Sir A. Garrod(Reynolds's System of Med.,1870,i,820),with some others,states that now and then acute rheumatism acts as an exciting cause of it,which appears to have been Fuller's(loc.cit.)view;he had repeatedly known it to commence apparently as a sequel of acute rheumatism. However,Ord(Brit.Med.Jour.,1880,i,158)met with a case in which the lesions of rheumatoid arthritis were present in a typical form in a patient who had mitral disease as a result of acute rheumatism,the arthritis being initiated as a continuation of the acute attack.It is not improbable that so common an affection as articular rheumatism should occur in the family or personal history of a patient the subject of rheumatoid arthritis;nasal catarrh and many other very common diseases must be frequent antecedents of the rheumatoid affection,yet are not causes of it.Vry much the same remark applies to the view that gout in the parents may transmit a tendency to rheumatoid arthritis in the offspring.The experience of medical men in this country in this matter is hardly reliable,owing to the great prevalence of gout therein. In Canada and many parts of the United States of America, however,while gout is a rare disease,rheumatoid arthritis is a common one,and observers there have not found an intimate relationship to obtain between the two affections.It is not intended to deny that when the children of rheumatic or gouty parents fail in health,owing to their inherited constitutional disease,they become liable to arthritis deformans,for that affection is predisposed to by all conditions enfeebling health.Many of the difficulties connected with this subject are reasonably met by Hutchinson's(Trans.Internat.Med.Cong., ii,95)doctrine that there exists a state of tissue-health which is transmissible by inheritance,which involves liability to inflammations of joints and fibrous structures,and upon this arthritic diathesis as a foundation may be built up,under the influence of special causes,a tendency to gout,rheumatism,or any of their various conbinations or modifications.He has shown that gout is often followed by rheumatoid arthritis,the lesions characteristic of both affections coexisting in the same joint.Charcot and Cornil(loc.cit.)had previously observed the same thing.Acute and perhaps chronic articular rheumatism have sometimes preceded arthritis deformans.If a predisposition,inherited or acquired,to rheumatoid arthritis exist,the occurrence of gouty or rheumatic irritation in the joints may suffice to induce the peculiar form of disturbance characteristic of the rheumatoid affection,just as the partial form is sometimes excited by traumatic influences.

SEX.

Rheumatoid arthritis is far more commonly experienced by women than men.Among five hundred cases collected by Sir A. Garrod(loc.cit.),only eighty-nine occurred amongst males;and the inequality in his findings would have been still more conspicuous,but for the fact that cases of the localised type were included,as well as those in which many joints were involved. This greater liability of the female sex is not confined to any particular period of life,but is observed even in the exceptional instances in which children are

affected;in old age it is least marked.We may take it, then that there is a group of conditions affecting the sexual functions and organs of women which appear tobe specially connected with the general peripheral form of rheumatoid arthritis.The disease follows pregnancy,and specially frequent pregnancies,protracted lactation,and various disorders of menstruation.The latter influence obtained in ten out of eleven instances of the disease met with in girls under eighteen by Fuller(loc.cit.).In the case of females the period of maximum liability to the disease corresponds approximately to the years following the period of the menopause,and common experience confirms the statistical proofs on this point.The influence of the menopause is often conspicuous even in the cases which date the onset of rheumatoid arthritis from an earlier period of life than the menopause;for the affection is apt to assume a fresh vigour at that time, and to resist treatment with an extraordinary tenacity. Todd noticed the coincidence of the affection with dysmenorrhoea.Ord(Brit.Med.Jour.,1880,i,151-153),in an able paper of grear originality,dwelt upon ovario-uterine disorder on irritation as a frequent active cause of the disease,having in his opinion met with thirty-three instances of the kind.The relationship between these various conditions of the functions and organs of generation and rheumatoid arthritis cannot be regarded as settled.Sir A. Garrod supposed that such conditions,by causing debility,predisposed to the articular disease.Todd, an ardent humoralist,held the nexus between the two to be unhealthy secretions of the uterus,leading to blood impurity;while Ord has ably defended Remark's view that a direct influence of the nervous system is the real link of relationship.It seems necessary to remark that mere coincidence may play a large part in the explanation of these cases.In seventeen at least of Ord's thirtythree cases the conditions stated by that author cannot safely be adduced as anything more;and it is probable that they would be found present in much the same proportion in any other chronic painful affection of the female sex.

ANTECEDENT AFFECTIONS.

Charcot,Cornil,and Sir A. Garrod all regard scrofula and tuberculosis as frequent frequently antecedents of rheumatoid arthritis;the first has several times seen white swelling in youth,followed by nodular rheumatism in later life;and Fuller found that twenty-three out of one hundred and nineteen victims of rheumatic gout had lost a parent or one or more brothers and sisters by consumption.Chlorosis has several times preceded rheumatoid arthritis.When the prevalence of scrofula,phthisis,and chlorosis is bornein mind,it will not appear strange that they should frequently be found amongst the antecedents of rheumatoid arthritis,without inferring any other relationship between them.It is especially such illnesses as leave behind them a lasting impairment of the general health thatmay be assigned an important place in the etiology of this disease. Influenza possesses this quality in a high degree,and no one who has paid attention to this subject can fail to have been struck by the frequency with which patients who have developed rheumatoid arthritis date the commencement of thie ailments from an attack of influenza

acquired during an epidemic of that disease.At the same time it must be remembered that,owing to the enormous prevalence of influenza during epidemic times,the relation may be more apparent than real;and there is,moreover, a widespread tendency to ascribe almost all affections to that cause.Gonorrhoeal rheumatism has also been occasionally known to precede rheumatoid arthritis;but Ord and Hutchinson(loc.cit.)are probably correct in regarding that malady as a variety of rheumatic gout.

COLD.

Long-continued exposure to wet and cold constitutes,it would seem,a not unimportant predisposing cause of rheumatoid arthritis,especially when associated with poverty and privation.It is for this,amongst many other reasons,that the disease is so frequently observed amongst the poor.When the disease has been fully established,fresh exposure to cold and damp is always attended by an increase of pain in the affected joints. Even years after the condition has passed away,a protracted residence in low,damp dwellings,deprived of the sun's rays and of a free circulation of air,is thought by many most favourable to the provocation of the disease.On the other hand,there are numerous observers who affirm that the influence of exposure to cold and damp have been greatly exaggerated.Certainly,those who suffer from the disease are extremely sensitive to changes in the weather,a fall of temperature or a spell of wet being often attended by a marked exacerbation of the articular pains;but even the patients themselves do not very often ascribe the commencement of their sufferings to such causes,or at any rate not so often as might be expected if they were very potent excitants.In this respect rheumatoid arthritis strikingly contrasts with true rheumatism;for an attack of the latter is very often indeed referred to cold and damp.The confusion of rheumatoid arthritis with true rheumatism is perhaps largely responsible for the widespread belief that it often dates from exposure,though it is impossible to deny that in some cases cold and damp,especially when prolonged have something to do with the production of the disease.

DEBILITATING INFLUENCES.

The old synonym for arthritis deformans, arthritis pauperum,would seem to imply that poverty and its concomitants may be at least frequent antecedents of the disease,as are other debilitatinf influences, such as night- watching,insufficient food,mental worry, grief,anxiety,etc.But it must be remembered that the disease is frequently observed in the affluent classes, who live in dry climates and warm houses,and want for nothing;so that the external conditions first mentioned are not essential causes of the disease,and many of them act merely as adjuvants.The dependence of the affection on a condition of lowered vitality is,in the opinion of some,specially illustrated by the effects of mental disturbance and depressing emotions.Anxiety, worry,and fatigue are frequent antecedents of the first symptom of rheumatoid arthritis,and during its course any unfavourable incidents of this kind are liable to be followed by an exacerbation of the articular disease. It has been affirmed that it is through the depressing influence of fright and other violent nervous shocks

that the connection between nervous depression and rheumatoid arthritis is frequently manifested. Some observers are very emphatic on this point, and patients themselves not infrequently refer the disease to such factors. Besides exciting the malady, worry and care also tend to increase the patient's sufferings where the affection is already established, so that depression of spirits, etc., make the pain experienced much more intense. Kohts (Berl. klin. Woch., 1873, p.304) reports two cases in which rheumatoid arthritis followed the shock of a shell bursting close to the patient; and Leyden (Klin. der Rückenmarkskr., 1876) tells us that in Strassburg not a few cases were produced by the bombardment of that city during the Franco-German War; but the general privation experienced at that time may have been operative.

DIET.

A liberal diet and the use of alcoholic beverages seem to exercise no influence in the production of rheumatoid arthritis; it is a disease that is benefited rather than caused by a generous supply of food. On the other hand, scarcity of nourishment, by leading to malnutrition, undoubtedly favours the development of the articular disease. In this respect rheumatoid arthritis stands in marked contrast to gout.

TRAUMATISM.

From a study of Charcot and Ord's cases (loc. cit.) it would seem that injury of a joint from a blow, a fracture, a whitlow, etc., sometimes induces a local rheumatoid arthritis, which may subsequently become multiple and involve several joints more or less symmetrically. The effect of local injury as an exciting cause of rheumatoid arthritis is most conspicious in the production of morbus coxae senilis rather than ordinary forms of the disease, though, as stated, it is undoubtedly true that injuries of the smaller articulations are sometimes followed by the characteristic deviations, deformities, and pains of rheumatoid arthritis.

Miscellaneous Factors.

Sterility in women is said to predispose to the occurrence of rheumatoid arthritis, as is also local disease of the generative organs; but here, again, there may be much in mere coincidence.

Finally, it may be noted that in a very large number of instances it is impossible to discover any assignable cause.

(B) PARTIAL, LOCALISED, OLIGO, OR MONO-ARTICULAR FORM.

The partial form of arthritis deformans presents certain peculiarities of causation; thus, it occurs chiefly in advanced life, and is then termed senile arthritis; much less frequently in the middle-aged, and very exceitionally in the very young.

Men are much more liable to it than women, and abundant statistical evidence proves.

It is chiefly this variety which follows injuries, blows, dislocations, pressure, etc., and the disease may then be limited to the injured joint and be mono-articular, or rarely, as in Ord's (loc.cit.) case, even general.

This mono-articular form appears to be sometimes induced by other local irritations of the articular structures than those following traumatic influences; and as foreign growths in joints and gouty irritation may respectively induce the lesions indicative of

rheumatoid arthritis,so,it is probable,may simple chronic rheumatism;and this may be the true relationship existing between the two affections. It is doubtful at present whether purely local irritation or injury of a joint can originate the alterations belonging to rheumatoid arthritis,that is, in the absence of all predisposition to that disease or of the arthritic diathesis.

Cold and dampness are admitted by numerous observers to the causes of the partial form,but the evidence on this point is not altogether satisfactory.It may be that chronic articular rheumatism is induced by the prolonged operation of damp cold,and that the prolonged rheumatic irritation,aggravated by constant use of the joint and by occasional violence,ultimately superinduces the profounder alterations characteristic of rheumatoid arthritis.It appears,according to some, highly probable that if the predisposition exist,any long-abiding irritation of a joint,whether the result of violence or disease,may ultimately occasion the lesions of the articulations characteristic of the malady under consideration.

(C)NODOSITIES.

Regarding the causation of Heberden's nodosities and their relation to other affections of the articulations,it may be noted that they obtain chiefly in advanced life,but do occur rarely in the young.

Sex appears to have some influence in their production,as they are probably somewhat more frequent in women than in men.

Although more often seen in the upper classes,the poor are not exempt from them,no doubt because they are specially exposed to slight but oft-recurring injuries of their digits,such traumatism being an exciting cause of the disease,especially when confined to a single joint.

The affection is sometimes hereditary;both it and the general or partial forms of rheumatoid arthritis may coexist in the same family,and even in the same individual.

The alterations in the joints are identical with those found in the general variety of rheumatoid arthritis,and exist without deposits of urate of soda. It resembles the general form of the malady just mentioned in its tendency to involve many symmetrical articulations at the same time,and the partial form in the rarity with which it extends beyond the joints first attacked.

While Heberden's nodosities,as Haygarth taught,do occur independently of gout and the gouty habit,Begbie (Contributions to Practical Medicine,1802,p.29),Duckworth(On Unequivocal Gouty Diseases, St.Barth.Hosp. Reps.,1880,Vol.xvi,p.190),and others believe that they are evidences of gout or the gouty dyscrasia.Cases have been reported in which middle-aged women have had these nodosities beginning on the last joint of the fingers,while they are the subjects of vesico-renal irritation and were passing free uric acid in the urine. Hutchinson(loc.cit.)has twice seen them in conbination with a peculiar insidious and painful inflammation of the iris and vitreous body,which occurs in the children of the gouty;yet such children have no deposits of lithates in their joints,not any lithiasis,nor acute paroxysms of true gout,and he considers that the last joint arthritis is to be regarded as in part gouty,

and in part a kind of articular chilblain.

It is maintained by certain writers that in some instances they are no doubt the hybrid offspring of an inherited tendency to both gout and arthritis deformans. "By inherited dispositions," says Paget (Lancet, 1882, 1017-1021), accumulating and combining or converging in definite proportions, new diseases may be developed and old ones be variously modified." Hutchinson writes to the same effect.

S Y M P T O M S.

C l i n i c a l V a r i e t i e s.

In considering the symptomatology of rheumatoid arthritis one cannot do better than follow Charcot's original division of the clinical types of the disease into the three following forms:

(1) General or Polyarticular or Multiple and Progressive Form.
(2) Partial or Oligo- or Mono-articular or Localised Form.
(3) Heberden's Nodosities.

The first-mentioned variety of the disease affects especially the smaller joints, but may soon include all. The second variety affects only one or two of the larger articulations, i.e., is central, not peripheral; while the third form occurs in the terminal articulations of the fingers.

It will be found convenient also to separately describe the peculiarities of the disease in children.

C l i n i c a l P h e n o m e n a.

(1) GENERAL OR POLYARTICULAR OR MULTIPLE AND PROGRESSIVE TYPE.

(a) Acute Form.

This variety of rheumatoid arthritis closely resembles the milder forms of acute articular rheumatism, or the best-marked examples of the subacute types of that disease. But it presents the following particulars, by which it may generally perhaps, but not always, be distinguished:

While the temperature, the thirst, the furring of the tongue, the frequency of the pulse, the articular pains and tenderness, etc., are less developed than in acute articular rheumatism, there is wanting the profuse and continued perspiration, the early involvement of the endocardium or pericardium in the inflammation, and the prompt prostration of the strength so commonly observed in that disease. On the other hand, while the rheumatoid affection may involve the larger joints, such as the knees and ankles and elbows and wrists, it almost certainly implicates the smaller joints of the fingers, and often of the toes. There is apt to be greater effusion into the synovial capsules - McLeod's capsular rheumatism - and into the synovial sheaths and bursae about the affected joints than in ordimary acute or

subacute rheumatism;further,the inflammation does not migrate from joint to joint,but obstainately persists in several of them,and more especially in the wrist and in the metacarpo-phalangeal joints of the index and middle finger,perhaps also in the ankles and in the matatarso-phalangeal articulation of the great toe.Instead of disappearing in from four to six weeks,the articular inflammation continues,although the pain may abate very much,and the capsules of the joints continue swollen and rather tense.The muscles of the extremities waste,and are the seat of painful reflex spasms which interfere with the movements of the joints;and although the patient is capable of moving about,and is free from all febrile disturbance,one or several of the joints remain permanently swollen,painful,and crippled.Perfect restoration of all the affected joints seldom,if ever, occursThis acute form is most frequently seen in young women twenty to thirty years of age;it has often occurred in connection with recent delivery or rapid childbearing,or lactiation,and sometimes after what was at first regarded as an attack of acute rheumatism occurring not lomg after labour.It has been observed in children,and is not uncommon after forty.These patients usually suffer in their general health - become weak, pale,depressed in spirits,and lose flesh.In several cases of this form marked intervals of improvement have occurred;the localdisease has ceased to progress,and tolerable comfort has been experienced,perhaps,till pregnancy,delivery,or lcatation again determined a fresh outbreak of the disease.Sometimes,however,this acute form steadily advances,and in a year or two establishes changes in the cartilaginous and osseous structures of the affected joints.

(b) Chronic Form.

This primary chronic form of rheumatoid arthritis is much more commonly observed than the type just described,although between it and the acute form there are many intermediate grades.For weeks or months the patient may experience numbness or formication and rheumatic pains in the limbs,perhaps with a sense of stiffness in the joints,especially felt after rest or the day after unusual fatigue.

Then one or more joints - most frequently the metacarpo-pharangeal of the fingers - become painful, swollen,tender when touched,and inordinately hot;these symptoms may subside under rest or treatment,and after weeks or months recur,either without unknown cause or from exposure,fatigue,or some impairment of health. Usually the original joint is again affected,but frequently one or two more of the same on the other hand suffer likewise.More or less complete remissions of the pain and local inflammation now tend to take place from time to time and alternate with exacerbations or fresh attacks of the local disturbance,and the disease extends, as it were,centripetally and more or less symmetrically to the wrists,then to the elbows,and then to the shoulders,or from the toes to the ankles and thence to the knees - although there is no invariable sequence of this kind - and next to the hands; the knees are specially liable to invasion.Of Haygarth's(loc.cit.) thirty-four cases,in two the knees alone suffered,and in all or nearly all the rest the hands,chiefly the

fingers, were probably involved. In Charcot's forty-five cases, the début took place in the small joints of the hands and feet twenty-nine times; in the hands, feet, and one large articulation, seven times; in one large joint, and later in the fingers, nine times. Even in this primary chronic form there is usually in the earlier stages some effusion into the joints; the soft parts of the articulation are thickened and swollen; obscure fluctuation in the smaller and very distinct fluctuation in the larger joints may be felt. The pain may be severe, especially at night, and during the exacerbations of the disease it varies greatly in its degree and persistency.

The position and shape of the joints are altered, partly by spasmodic retraction of the muscles, and more or less by the effusion into the capsules and adjacent bursae and sheaths, and the thickening of the soft parts covering the articulations. As the disease progresses, future deformities ensue from the growth of new bone around the heads of the bones, the absorption of the articular cartilage, the development of masses of cartilage in the hypertrophied synovial processes and beneath the synovial membrane at the margin of the bones; the relaxation of the articular ligaments; and the displacements and subluxations of the unshapely bones composing the joint. The great wasting of the muscles of the member affected has some share in producing its unnatural appearance.

In the advanced stage there is more or less abiding pain, soreness, and siffness in the affected articulations, violent cramps are experienced in the course of the adjacent muscles, and pains either along the nerves or vaguely down the limbs. Creakings or cracklings are to be heard, and grating is to be felt during the movements of the joints; these movements become more and more restricted, so that an immobility almost equal to that of true bony ankylosis is established, this result seldom occurring except amongst the carpal, tarsal, tibio-tarsal, and the vertebral articulations. Interlocking of the osteophytes formed on and around the articular surfaces, and in other cases union of these surfaces by the interposition of newly-formed tissue, produce a spurious ankylosis destructive of the articular functions.

In the very advanced stages the feet, ankles, and legs are often considerably enlarged and the integument thickened by a chronic oedematous infiltration, or the bones or soft parts are atrophied and the integument is pale, smooth, and attenuated, resembling parchment or the condition seen in certain stages of scleroderma and tightly drawn over the wasted and rigid fingers.

This primary chronic form is especially apt to progress steadily for years and years, the joints earliest affected becoming gradually more distorted and crippled, and fresh joints becoming invaded until there may hardly remain a single sound articulation in the limbs, or even in the body; and at length the patient may be unable to feed himself or masticate or raise his chin from his sternum or rotate his head or stand upright on the ground. As a rule, the pain is less in the older cases. The trophic disturbances of the skin are manifested in its becoming dry, smooth, and glossy, especially over the swollen joints, and it is sometimes pigmented or, as Spender (Brit. Med. Jour., 1891, May 30)

calls it,"freckled"there.Anders(Practice of Med.,p.390) reports three personal cases of onychia,a condition also observed by many others.He also refers to the free perspiration in advanced cases,which very few others seem to have observed.Bedsores are less common than would be supposed with so much emaciation and tendency to trophic disorders.

It must be remembered that the lesions of the several articulations,being largely the outcome of muscular contraction,observe certain general types,which,however are not peculiar to the disease,but occur in various affections of the nerve centres,involving paralysis or spasm,or both.Charcot(loc.cit.)has carefully described those seen in the hands,and,according to his observations,confirmed by innumerable others,the predominant features of the hand in rheumatoid arthritis are the following:

The first phalanx of the fingers is either flexed upon the metacarpus or extended,and the terminal phalanx in like manner is either markedly flexed or extended upon the second,or these two phalanges are maintained in a straight line,while the first phalanx is,as usual,decidedly flexed upon the metacarpus(Lectures on Senile Diseases,Syd.Soc.,1881,and Figs.1 and 2,Pl.II., illustrating these deformities in the hand).In all these varieties the hand is pronated;there is a great tendency to deviation of the fingers toward the ulnar border of the hand,although sometimes the deformed fingers stand out,not unlike a bunch of parsnips.The thumb escapes longer than the other fingers,and flexion,rarely extension,is observed in its metacarpo-phalangeal articulation.The great toe,enlarged at the metacarpo-phalangeal joint,is usually drawn to the outer border of the foot,across and above,but seldom below,the other toes, and the foot is usually abducted and flattened,the prominent internal border resting on the ground.The wrist, elbow,and knee-joints are generally flexed;the distal ends of the ulna and radius,more or less enlarged,project backwards;the semi-flexed tibia is drawn backward on the femur and rotated outward,thus rendering the internal condyle of the femur prominent and displacing the patella toward the external condyle,and foreign bodies may frequently be felt in the enlarged knee- and elbow-joints.Finally,the extremities of the affected bones will,as a rule,be found enlarged and misshapen,and upon them will be felt nodosities,rims,tips,ridges,and stalactiform osseous new growths.

Abarticular Phenomena.

In the chronic form of the disease variation is exhibited in various individuals,and there is no characteristic disturbance of the functions,such as obtains in chronic gout.

Temperature.- Fever is always present in the acute type of the disease,but it is irregular and not very high.Hectic fever is sometimes observed,showing a normal registration in the early part of the day and a rise of two or three degrees toward evening.The variations in the amount of febrile phenomena seem sometimes to have no connection with the articular processes.

Urine.- The urine is not characteristically affected in rheumatoid arthritis.There is a diminution in the amount of the earthy phosphates,and there is a

great variability in the quantity of urea and uric acid that are contained in the urinary excretion. Albumin, in the absence of intercurrent renal disease, is not present. Marrot (Contribution to the Study of Articular Rheumatism: Examination of the Urine and of the Blood, Paris, 1879, p. 42) says that he found both urice acid and urea notably below the normal quantity in the urine, although the former increased notably under baths and high temperature.

Skin.- It was long ago noticed that the skin covering the affected joints, especially when the fingers are involved, frequently exhibit dystrophic changes, of which the phenomenon of "glossy skin" was one of the most usually seen. The condition was first commented upon by Spender (Proc. Med. Soc., 1888, xi, p. 209; Osteo-arthritis, 1889; Brit. Med. Jour., May 30, 1891), who went fully into the question of pigmentation of the skin in the neighbourhood of the affected articulations, and considered the development of these frecklings as the most pathognomonic of all the clinical signs and present in about two-thirds of all the typical cases of rheumatoid arthritis. His observations have since been confirmed by others. These pigmented spots are sometimes almost black in colour, and are to be found in the neighbourhood of the implicated joints. In many instances their appearance has been directly associated with that of the joint disease; but it must be borne in mind that such spots are not infrequently to be seen in the case of individuals who are not suffering from rheumatoid arthritis, but who have reached the period of life at which the affection is most commonly observed, and that, even when the two are associated, the patients will often affirm that the pigmentation has been there as long as they can remember. It would seem, then, that their diagnostic importance has been exaggerated. The glossiness of the skin is very often observed; sometimes the integument in the vicinity of the affected articulations has merely an unusually shiny appearance, and seems to be unduly thin; but occasionally the typical pink tint and glossy surface of cutaneous dystrophy is encountered. Indeed, trophic ulcers have in exceptional instances been observed. Perspiration has been observed sometimes, but is obviously not peculiar to this disease. Oedema of the legs may occur, and in the absence, too, of any visceral lesions to which it can be referred.

Nails.- The nails frequently become brittle and longitudinally curved as in gouty subjects. Dystrophic lesions may be seen in rare instances. Ulceration of the fingers and atrophy of their pulp are not uncommon features.

General Health.- The tongue may be clean, the pulse tranquil, the appetite and digestion satisfactory, and the urine normal or perhaps of low density. Fuller (loc. cit.), however, says that "more generally the complexion is sallow and the skin sluggish, and evidence of mischief is furnished by the yellowishness of the conjunctivae, constipation of the bowels, a pale and unhealthy character of the dejections, excessive flatulence after meals, turbidity of the urine, and fulness of the pulse." My own experience hardly harmonises with this, and I have seen individuals suffering from the general and partial form in the enjoyment of excellent general health. Should, however, the disease develop in a person

the subject of menorrhagia or other urine disorder, or of repeated child-bearing, or after prolonged mental anxiety, some disturbance of the general health fairly referable to such disturbing conditions may be certainly looked for. Anaemia, dyspepsia, and failing health in the advanced stages are the results of the prolonged suffering and confinement.

Blood.- Sufferers from rheumatoid arthritis are nearly always anaemic, the condition being especially marked in the very acute cases and in the chronic forms of long standing. Purpuric spots are sometimes seen, but haemorrhages are exceedingly rare.

Heart.- Such abarticular affections as those of the heart have occasionally been observed in persons suffering with rheumatoid arthritis, but many even of those authors who regard the disease as a form of rheumatism speak of these affections as coincidences, and not as essential manifestations of the disease. Charcot and Besnier (loc.cit.), however, maintain the latter to be their true relation to the articular affection which they regard as chronic rheumatism. The two authors just mentioned allege that all the visceral localisations that occur in acute articular rheumatism may obtain in the nodular form, but that such localisations are infinitely less frequent and serious than in the acute, subacute, or simple chronic forms of articular rheumatism - that endocarditis and pericarditis undoubtedly do occur in nodular rheumatism, and appear especially when there is an excerbation of the disease and where there is some approach to the acute stage. As Charcot has adduced these cardiac affections in proof of the rheumatic nature of rheumatoid arthritis, it is deserving of mention that he had personally met with but two instances of endocarditis and five of pericarditis, four of the latter having been discovered not during life, but in nine autopsies, and that he cites only eight other cases of endocarditis or pericarditis which had been eother published or reported to him. He admits, moreover, that there had generally been in these cases, at some former period, an attack of acute rheumatism. Besnier, Homolle, Malherbe, Vidal, and Colombel, in their articles upon the disease under consideration, so not cite a single case in which they have seen cardiac disease in rheumatoid arthritis. On the other hand, McLeod, Sir A. Garrod, Fuller, Flint, Pye-Smith, and others either deny or ignore the occurrence of cardiac disease as a manifestation or complication of this disease. My own personal experience coincides with that of those writers last mentioned. If those instances be included in which a former attack of acute rheumatism might be adduced in explanation of the supervention of heart disease, but few cases will remain to suggest that rheumatoid arthritis may develop endocarditis or pericarditis; and when it is borne in mind that in several ways the cardiac affections may have arisen as mere coincidences of the rheumatoid affection, it is well to wait for further evidence before accepting as proved the occurrence of cardiac affections as local manifestations of rheumatoid arthritis. The form of the disease in which acute carditis has occurred may be rather that of true articular rheumatism of a very subacute type.

Respiratory System.- Nor is the evidence at all

satisfactory in favour of any special tendency to the following affections, much less of their being local manifestations of rheumatoid arthritis, namely, pleurisy (McLeod, Fuller), astham (Charcot), and chronic laryngitis (Garrod), as well as such intercurrent affections as bronchitis and pulmonary tunerculosis.

Kidneys.- The above remark holds true also as regards the occurrence of renal disease, which sometimes complicates arthritis deformans, and is among the more frequent causes of death in these cases.

Eye.- Inflammation of the comjunctiva, iris, and sclerotic capsule of the eye is occasionally observedm but in a certain proportion of such cases may be referred to gout or gonorrhoeal rheumatism, which is not infrequently accompanied by ocular troubles.

Ear.- In a considerable number of cases deafness results from rheumatoid arthritis involving the articulations of the small bones within the ear.

Nervous Manifestations.- Gowers (Dis. of the Nervous System, i, p. 381) says that the trophic muscular wasting, which is as a symptom secondary only to the articular lesions, is often accompanied by by increased myotatic irritability. The atrophy affects the extensors in greater degree than the flexors, and hence flexion is more common than extension, but extension may easily be produced by osteophytes. Other clinicians find that the reflexes vary, and in so far as they can be tested in rigid limbs may be either much increased or diminished. Clonic spasm in extension of a limb is now and then observed. Very early in the course of the affection numbness and tingling of the hands and feet may be experienced, even before the joints appear to be involved. The pains experienced by the subjects of rheumatoid arthritis may be of various kinds. That arising from the joint affection is of variable severity, and in some cases of the most chronic type is so slight that the patients themselves treat it with more or less contempt, being worried more by the arthritic deformities than it. But is is often severe, and may cause agonising torture. The pain is increased by movement, and by the warmth of the bedclothes also. We have already seen that changes of the weather and mental emotion may make it worse; and the twinges are usually of a gnawing or burning kind. As the disease progresses the pain is less complained of, and it is fortunate for the patient that this is so. Again, pains may be experienced in other situations than the affected articulations; thus there may be a neuralgic pain in the ball of the thumb or on the inner side of the wrist, which Spender regarded as almost a pathognomonic signs of the disease. The bones themselves may sometimesbecome exceedingly painful, apart from the articular disease. Painful spasms on the periarticular muscles may cause the patient great suffering; these, like cramps from other causes, are of sudden onset, are apt to cease as suddenly as they began. The spasmodic contractions of the muscles on the joints greatly aggravate the painful condition of the latter. Furthermore there may be radiating pains due to implication of the nerve-roots, but only when the vertebral column is diseased. These pains are radiating and burning, follow the course of a spinal nerve, and associated with spondulitis, and are apparently rather frequently

observed.They now and then follow the course of the occipital nerves,or may manifest themselves as intense brachial or intercostal neuralgia.

Rheumatic Nodules.- Occasionally fibrous nodules may be discovered amongst the muscles of the upper extremities,but these must not be mistaken for the nodules of articular rheumatism to which latter affection they are peculiar.Stephen Mackenzie has seen them in one instance of tertiary syphilis,the patient not having had arthritis,chorea,or rheumatism.It may be that when they occur there has been an antecedent attack of rheumatism to account for their presence;or the condition may not be a true rheumatoid arthritis but a simulating affection;but it remains for the future to determine their real nature.

(2) PARTIAL OR OLIGO-ARTICULAR OR MONO-ARTICULAR OR LOCALISED TYPE.

This form of rheumatoid arthritis,like that above described,is usually a primarily chronic affection,insidious in its invasion and slow in its course.It is chiefly observed in old people,especially men,when it is called senile arthritis,affects frequently a single joint,and chiefly the hip,but occasionally the knee, shoulder,or spinal column,either as a consequence of special injury or of the wear and tear of life,or exposure to cold and wet,or even of what seemed to be simple acute or subacute articular rheumatism or gonorrhoeal rheumatism.When not the result of injury,two or three joints may suffer,both hips or knees,or hip and some of the vertebrae,hip,knee,and ankle of the same limb,and so on.Even in those cases in which the disease for a long time is confined to a single joint and may have been caused by an injury(Ord,- Brit.Med. Jour.,1881,i,158),other joints are finally apt to become affected,often in symmetrical order.So that it may seem almost general,or at least polyarticular,just as the converse sometimes happens in the general rheumatoid arthritis of long standing,where the affection becomes greatly aggravated in one joint and produces great deformity and destruction of it,the others remaining in statu quo.

The clinical features of localised rheumatoid arthritis closely resemble those of the general variety detailed above;but there is usually in the early stages less heat,tenderness,and dwelling of the affected articulation.The pain is less acute,but more abiding,and, with the exception of more or less stiffness or impeded movement in the joint,it may be the only sign of disease present,so that at this stage of the affection it may be taken for simple chronic articular rheumatism.But the disease persists;the voluntary movements become more painful and difficult;slight exercise of the joint is followed promptly by fatigue and aggravation of the pain,and yet the articular surfaces may be pressed together,and flexion and extension be practised,without causing much suffering.Slowly and continuously alterations take place in the affected articulation;with but little heat or redness it enlarges steadily,the soft parts becoming infiltrated and thickened,or effusion taking place into the capsule;the articular surfaces become irregularly depressed by the growth of osteo-cartilaginous rings,osseous nodosities,and stalactiform

processes upon them,and these irregularities,together with one or several loose bodies,may be felt in the joint.The enlargement of the articulation becomes more apparent,owing to the wasting of the muscles of the limb;its movements become more and more restricted and difficult,although perhaps not more painful,and are attended with creakings and gratings perceptible to the ear and hand;and at last nearly all the movements of the joint may be prevented by the alterations in the shape of the epiphyses,or by the interlocking of the osseous outgrowths,or in rare cases by actual union of the bones. This form constitutes,par excellence,arthritis deformans. In many instances there is little effusion throughout the process,notwithstanding the great deformity in progress;hence the term dry arthritis.Even the partial form is sometimes more active in its invasion,as when it very rarely succeeds acute or subacute articular or gonorrhoeal rheumatism,or,more frequently,ensues after traumatism.

The partial form of rheumatoid arthritis is generally very protracted in its course;it may last for ten or a score of years.Exacerbations of the affection occur from time to time,in the intervals of which,although the implicated articulations are seriously crippled,the patient may be free from pain.The malady is not in itself fatal;the patient may live to a great age and die of some intercurrent disease,such as pneumonia,apoplexy, or senile decay.

A description of the features presented by partial rheumatoid arthritis affecting the hip,the shoulder,and other joints rather appertains to works on surgery,and only a glance at the evidences of the disease in the hip and in the vertebral column will here be given.

The affection in connection with the hip is called morbus coxae senilis,and its commencement is indicated by a dull gnawing pain in the neighbourhood of the joint and in the knee.Rotation of the head of the femur in the acetabulum becomes more and more difficult,and the affected thigh can only with difficulty be raised and crossed over the other.Stiffness of the tissues around the hip-joint increases,though pressure upon the trochanter is not particularly painful,and the patient continues to more about with some degree of freedom.As the disease advances,however,the muscles of the hip undergo atrophy,and in many instances there is similar wasting of the muscles of the thigh.The knee-jerk is somewhat increased;the head of the femur is gradually eroded and absorbed,so that the limb becomes considerably shortened;sometimes large cycts are developed outside of the joint,often without any apparent connection with the articular cavity.In advanced stages of the disease,after destruction of the articular surfaces,a grating sound is audible when the thigh is moved upon the pelvis.

When the cervical vertebrae are implicated - spondylitis deformans - the power of rotating the head from side to side is usually preserved and is attended with a crackling noise,while the rest of the cervical region is stiff and the head cannot be bent forward; when the dorsal or lumbar vertebrae suffer the back becomes bent,the patient stoops greatly and cannot stand erect,and his body is shortened and more or less twisted.A careful examination will discover not only

the great rigidity of the spine,and as it were fusion en masse of the joints,but in these persons the bony outgrowths may be felt.Occasionally the alteration in the vertebrae by compressing the cord or its membranes, or the spinal nerves and glanglia,may produce neuralgic pains in the cervical,dorsal,lumbar,or sciatic nerves, wasting of the muscles,more or less paralysis,and even vasomotor troubles.

Sometimes the mono-articular form of rheumatoid arthritis is merged in a subsequent development of polyarthritis deformans of the same nature,but the affection remains localised in the hip-, shoulder-, or knee-joint in the majority of instances.

(3) HEBERDEN'S NODES.

Undoubtedlythese are sometimes the effect of rheumatoid arthritis,implicating chiefly,and often solely,the distal joints of the fingers,where it slowly forms too little hard nodules about the size of dried peas upon the sides of the articulations.These are notably changed,and their movements impaired,but pain is seldom experienced,and were it not for the deviation of the finger to one side or the knob-like excrescences upon the joints - appearances which much disfigure the hand - patients would not speak of their ailment. In many cases these alterations likewise involve,but in a minimum degree,the first phalangeal articulations,and less frequently the metacarpo-phalangeal,and even some of the larger joints - the wrist,knee,or hip,etc.Like the other varieties of rheumatoid arthritis,this form occasionally has a more active invasion than is above mentioned,and may be attended by local pain,heat,and redness,or such symptoms may occur as exacerbations of the chronic affection.These nodosities may be preceded by gout,or the latter may precede the former by many years;they are often accompanied by asthma,migraine, neuralgia,especially of the sciatic nerve,and muscular rheumatism;and these manifestations may,it is said,alternate with exacerbations of the disease.Cystic enlargements or herniae of the capsules may be present,filled with clear fluid.Radial deflexion of the joints may occur in opposition to the ulnar deflexion which is typical of the involvement of the entire fingers and hand.As in gout,the thumbs usually escape.The nodosities are not associated with urate deposit;and,although such has been maintained,they bear no relation with gout.

RHEUMATOID ARTHRITIS IN CHILDREN.

Arthritis deformans is not common in children,but is more often encountered than tophaceous gout.In them it seems to be more dependent on bad hygiene than in adults.Poor and insufficient food and foul air or exposure to cold and damp are apt to develop it.It runs a rather more acute course as compared with the disease in adults,the sexes are more equally represented,and in some cases it has been associated with goitre, presenting a rapid pulse,tremors,and cutaneous pigmentation,as the cases reported by Spender(loc.cit.) and Diamanthberger(On Nodular Rheumatism in Children,These de Paris,1891) show.From evidence advanced by various observers it would seem that,although a disease ident-

ical with rheumatoid arthritis of adults does occur in children, certain cases of articular disease, usually regarded as the former condition, are reality not of that nature, but constitute a class by themselves. There are no special clinical phenomena to be observed distinguishing the affection from the adult disease. The malady is associated with the smaller joints of the extremities, the form found in the younger adult patients, as contrasted with that affecting the larger joints, which is, in the vast majority of instances, a senile change. It begins probably in the same way with fugitive articular pains, then stiffness, especially after the joints have not been moved for some time, as first thing in the morning. Then comes a little swelling of the articular ends of the bones, with perhaps slight tenderness. Next follows some displacement, owing to altered shape, the formation of nodules, excrescences, and erosion of cartilages, with grating sounds when the articulation is put in motion. The enlargement of the joints is, then, progressive; and it is associated with general enlargement of the lymphatic glands and of the spleen. The affection nearly always commences before the second dentition, and certain writers say that girls are more affected than boys, though others deny this and maintain an equality of the sexes as regards incidence; it differs clinically in the absence of bony changes in the joints and in the presence of the adenopathy above named. The most striking difference in the morbid anatomy is the absence of fibrillation of cartilage even in advanced cases, the absence of bony lipping or grating, or any osteophytic change, and the fusiform enlargement of the joints. The incidence of the articular affection has been adduced by certain authors as a further point of difference from the adult disease, the knees and wrists being first attacked, instead of the small joints and fingers, as in rheumatoid arthritis of the ordinary type; and another characteristic is said to be the occurrence of adherent pericardium in rare instances, with no changes, or at most a doubtful thickening of the mitral valve. Still (Med.-Chir. Trans., 1896), who bases his assertions upon a study of twenty-two cases, says that such variations cannot be merely due to difference in age, for a disease exactly corresponding to the rheumatoid arthritis of adults also occurs in quite young children. The condition is further differentiated from the rare form of chronic rheumatism described by Jaccoud as polyarthrite déformante; in that affection there is evidence of genuine rheumatism, such as nodules, and organic valvular disease, while on the other hand there is absence of enlargement of the spleen and lymphatic glands; Jaccoud's disease, again, is distinguished from the true rheumatoid arthritis of children by the absence of bony grating and thickening, such thickening as exists being periarticular. It is consequently suggested that under the term rheumatoid arthritis three conditions have, in the case of children, been grouped together which are actually separate affections; these are - the form in which there is no cartilaginous or osseous change, but glandular and splenic enlargement; true rheumatoid arthritis, as in grown-up persons; and an exceedingly rare form, probably identical with the polyarthrite déformante of Jaccoud. The glandular enlargement has been described by

several writers, including Bannatyne, Wohlmann, and Blaxall (Lancet, April 25, 1896), and Chauffard and Ramond (Rev. de Med., May, 1896), as occurring in adults in an acute form of rheumatoid arthritis, which they distinguish as infective arthritis. In the case of children however, the most striking feature of the disease is the remarkable wasting of the muscles in relation to the articulation, and also a thinning and glossiness of the skin over the affected area. The muscular wasting is best seen in the hands, which are most often the first parts affected, when the atrophy of the extensors, together with the deflection of the phalanges to the ulnar or less commonly to the radial side, and the flexing of the fingers, gives the hand the peculiar claw-like appearance which is pathognomonic of the disease.

DIAGNOSIS.

There are various conditions from which rheumatoid arthritis may require differentiation, of which the following are the most important:

RHEUMATISM.

It is sometimes by no means easy, it may be impossible perhaps, to differentiate either the acute or the chronic form of rheumatoid arthritis from subacute or chronic articular rheumatism respectively before the characteristic deformities of the former affections have appeared. Acute rheumatoid arthritis, which is comparatively rare, may be said to exist, rather than subacute articular rheumatism, if the disease affect early and chiefly the smaller joints of the hands and feet alone or along with some of the larger articulations, especially the sterno-clavicular or the tempero-maxillary; if the effusion into the joints be abundant; if the inflammation persist in the articulations first involved, notwithstanding the invasion of other joints; if the heart escape; if the patient be a female who is constitutionally delicate, or has borne children rapidly, or is the subject of disordered menstruation, or has been attacked soon after childbirth or during lactation; finally, if, on cessation of the attack, one or more of the joints remain swollen and permanently enlarged and impaired in function. The coexistence of iritis, or a history of a previous attack of that disease not attributable to syphilis or gout, would make the presumption more certain.

Pretty much the same points may be taken into consideration in distinguishing chronic general or polyarticular rheumatoid arthritis from chronic articular rheumatism, with the following qualifications: endocarditis or pericarditis is not of frequent occurrence in chronic rheumatism, so that this distinction is not available, and chronic articular rheumatism of long standing does sometimes impair the movements of the joints, and even produce slight alterations in them. However, it does not, as a rule, involve so many joints

as rheumatoid arthritis;it is less symmetrical in its distribution,and much less prone to implicate the sterno-clavicular,the tempero-maxillary,or the vertebral articulations.Nor does it cause removal of the articular cartilage,enlargement of the heads of the bones,and the formation of osteophytes around them,and of loose bodies in the articulations,together with marked deformities and luxations of the joints.The simple rheumatic nature of the case would be strongly indicated by a history of a remote or recent attack of acute articular rheumatism or of chorea,or the presence of chronic valvular disease.

If it be difficult to distinguish the general form of rheumatoid arthritis from chronic articular rheumatism,it is much less easy to do so in the case of the partial type before the characteristic alterations of the joints have been developed,more especially as it is sometimes a consequence of gouty irritation and probably of chronic rheumatism.Chronic inflammation of an articulation following a traumatic cause,and persisting obstinately in the injured joint is probably rheumatic, if not periarthritic,gouty,or tuberculous.

MONO-ARTICULAR ARTHRITIS OF THE SHOULDER.

An affection of the shoulder frequently occurs which resembles in many respects rheumatoid arthritis, and has been graphically depicted by Duplay(Arch.Gén. de Méd.,Nov.,1872,pp.512-542) and others.Anders(loc. cit.)has seen five cases,and Osler(loc.cit.)refers to others.It is very important to be able to distinguish this mono-articular arthritis of the shoulder,with its neuritis and local muscular atrophy,from the localised type of arthritis deformans when it affects this joint. The affection generally follows an injury,such as contusion,sprain,etc.,of the articulation named,but may be spontaneous;it is unattended by swelling or deformity. Its early symptoms are pain on pressure a little below the outer border of the acromion,and especially behind it and at the coracoid process,also about the insertion of the deltoid and below the acromion during movements of the joint,especially when the arm is raised from the side or rotated inwardly;early restriction of these movements,which increases till a fibrous ankylosis becomes established and scapula and humerus move together as one piece,motion between these bones no longer existing,and forcible attempts to produce it giving great pain,and sometimes producing crepitus in or about the articulation;sometimes early numbness and pain down the member to the hand,in the course of the ulnar,internal cutaneous,or the radial nerve;vicious and painful semiflexion of the elbow;after a time wasting of the groupof muscles which move the shoulder-joint.Although usually mono-articular and of traumatic origin,in rare instances it affects first one and then the other shoulder in the absence of any known injury,and beginning like a neuritis or a neuralgia of the scapulo-humeral nerves.Duplay,however,regarded it as a peri-arthritis.It may be distinguished from the rheumatoid arthritis by the absence of effusion into or enlargement of the articulation,and of deformity of the bones;by the curability of the disease;and by the early restriction of the movements and the development of adhesions which fix the joint.The morbid anatomy is wholly

different in the two diseases, for in it the bones and cartilages are not involved, but only the capsule and ligaments; furthermore, the patients recover after a subacute and painful attack.

TABES DORSALIS.

Charcot's (Lectures on the Nervous System., Syd.Soc.Ed., 1877; Arch.de Phys., 1868, i, p.161; ibid., 1869, xi) disease, which is the name given to that form of locomotor ataxia which presents destructive articular lesions somewhat resembling those of rheumatoid arthirits of the mono-articular form is distinguished from the latter by its sudden invasion, often without pain or fever; the prompt development of a general and often enormous tumefaction of the entire member, with copious effusion into the joint; the early destruction of the articular cartilages, the rapid wearing away of the heads of the bones, and the proneness to spontaneous fracture of their brittle shafts; the prompt absorption of the articular effusion, followed by a relaxed state of the ligaments and a facility of dislocation; the early occurrence of the articular affection, when motor incoordination is scarcely developed, and its frequent association with the crises of ataxia or the presence of some of the other symptoms of the disease. In such cases the Roentgen photogram is of great value.

PROGRESSIVE MUSCULAR ATROPHY.

Articular lesions closely resembling those seen in tabes dorsalis are now and then observed in the early stages of progressive muscular atrophy, which latter affection can be easily diagnosed from the former by the characteristic abarticular symptoms, and also by the general history and study of the case.

SYRINGOMYELIA.

This affection may sometimes exhibit articular lesions similar to those of rheumatoid arthritism, which may last, according to Beevor and Lunn (Trans.Clin. Soc., 1894, xxxvii, p.209); but the other symptoms of spinal-cord disease are always present to establish the true nature of the symptoms observed.

GOUT.

I have not infrequently experienced great difficulty in making up my mind as to whether a given case is an example of rheumatoid arthritis or of chronic gout; the discrimination is obviously important, especially as the treatment for these affections, as well as the prognosis, is radically different. To the distinguishing points presently to be enumerated there are occasional exceptions, as such affirmations can naturally only include average or typical cases. It is, however, the irregular or obscure cases which are deceiving, but in these several of the symptoms can be slected by which a positive diagnosis can be made. Arthritis deformans, while it most often begins in the hand, and is usually symmetrical and bilateral, contrasts with gout which commonly commences in the lower extremities, and especially in the metatarsal joint of the great toe, and of one foot only. Chronic gout is far more frequently preceded by attacks of acute gout than chronic rheumatoid arthritis is by the acute form of that affection; a history of inherited predisposition, of indulgence in the use of wine, ale, porter, and of animal food, of deficient bodily exercise, with perhaps great mental occupation or anxiety, of recurring gouty dyspepsia, or of

a tendency to lithiasis,would indicate gout;while the absence of these and a history of frequent exposure to cold and wet,of injury to the joint,and of previous exhausting disease or drain,of impaired health,debility, or poverty,would strongly imply rheumatoid arthritis. Gout is especially observed in males over thirty,and very rarely in children;generally rheumatoid arthritis is chiefly a disease of females during menstrual life, and occasionally occurs in children of either sex.The partial form is,like gout,chiefly a disease of males, but occurs generally at a more advanced age than gout. Even chronic gout is more or less paroxysmal,with distinct intermissions;chronic rheumatoid arthritis is more or less abiding and progressive,with only remissions in its course and severity;the former is frequently associated with chronic renal disease,the latter is not.The uratic deposits about the articulations in gout appear as more or less rounded or ovoid swellings in the close vicinity of the joints,but not observing their exact level or their general form;softish when recent,they never acquire a bony hardness,and are nearly always capable of slight lateral movement.The skin covering them is frequently stretched and glossy,and may exhibit white spots of urate of soda.The articular nodosities in chronic rheumatoid arthritic are actual osseous enlargements of,or outgrowths from,the articular surfaces, forming part of them,immovable and conserving more or less their form.The skin covering the nodosities is not glossy or dotted with chalk-like specks.The several types of deformity of the fingers previously described, and mainly produced in rheumatoid arthritis by muscular contractions and altered shape of the articular surfaces,are not seen in gout.Finally,if chalk-like concretions are visible in the ears,joints or finger-tips,or if the blood contain uric acid,gout is present.

SYPHILIS.

A chronic arthritis may be observed in adults amongst the very late manifestations of syphilis.It is usually mono-articular,affects the larger joints,especially the knee,and may originate either in the synovial membrane or in the bone and periosteum.In syphilitic synovitis the history of the case,the existence occasionally of soft gummy tumours in the periarticular tissues and of hydrarthrosis,the trivial degree of pain and tenderness,the insidious invasion and chronic course of the disease,and its prompt relief by specific remedies, will make a positive diagnosis possible - the more so should there be a Wassermann reaction obtainable,and the spironema pallida be found.

SCIATICA.

From this affection morbus coxae senilis may be distinguished by the absence of tenderness,though pain may be present along the sciatic nerve,the limited rotation of the diseased hip,the increase in pain produced by crowding of the femur against the joint, and the difficulty or impossibility of crossing the diseased leg over the sound one while in a sitting posture.There is also flattening of the glutei,and on the affected side an increase of the knee-jerk will be observed.

HAEMOPHILIA.

This disease occasionally presents articular lesions analogous to those of rheumatoid arthritis,

which,however,are often accompanied by effusions of blood into the joints and fibrous adhesions(Bowlby,-St.Barth.Hosp.Reps.,1890,xxvi,p.77),which are not to be seen in the latter affection.

TUBERCULOSIS.

Tubercular inflammation of the joints somewhat resembles rheumatoid arthritis,but it does not produce the characteristic deformities of that disease.

In the case of C H I L D R E N it is not infrequently most difficult to distinguish the affection under consideration from the more chronic or subacute forms of true rheumatism until the characteristic deformities occur;indeed,in not a few instances rheumatoid arthritis would appear to be a later development or sequel of the acute form.But the real nature of the malady become apparent when the enlargement of the joints,the crepitus on movement,the wasting of the muscles,the thinned glossy skin,and the distortion of the fingers are observed.

P R O G N O S I S.

The prognosis of rheumatoid arthritis as regards life is good,but for ultimate cure it is hopeless, though of late the prospects of cure appear to be somewhat more promising,especially when modern treatment is applied at an early stage of the disease,before the pathological changesin the articulations and the atrophy of the muscles have progressed too far.Marshall (New York Med.Jour.,June 19,1897)says that the prognosis of this disease is,in general uncertain;many cases progress to a condition of helpless deformity and consequent helplessness,although the general health may not be seriously inpaired,and death may take place from some intercurrent affection;in others the deformity and impairment of function are limited in degree;the prognosis is,he adds,not unfavourable in those cases in which the condition receives early recognition before the permanent lesions have developed.The course of the generalised or polyarticular form of rheumatoid arthritis varies much more thanis generally supposed,and the affection must not be regarded as necessarily progressive and incurable.When it occurs in young persons,and in children more especially,although it may suffer exacerbations and remissions for a few years,yet arrest of the disease and recovery of the functions of the joints, sometimes with very little deformity,now and then take place under suitable managementOne sometimes finds in younger female patients,while bearing children rapidly andnursing them that they have had the disease in their hands and wrists or hands alone;that exacerbations have recurred during subsequent lcatations,and yet the disease has either become arrested or progressed very slowly and at long intervals.But these are all exceptional cases,and the tendency of both the polyarticular and of the mono-articular forms is to progress,and,

either steadily or at intervals and by recurring attacks, to permanently deform the joints and impair their movements. Even under these circumstances, however, the patients may suffer little pain, unless one endeavours to move their joints. But, though the affection cannot be regarded as curable under the exhibition of drugs, very much can frequently be done, especially in the polyarticular form, to relieve the suffering and to retard, if not arrest, the progress of the disease, and even to restore sometimes very considerably the functions of the joints. Neither of these forms of rheumatoid arthritis can be said to be dangerous to life, and they often half a score or a score of years without seriously affecting the patient's general health. Heberden's nodosities cannot be cured; this does not, in a sense, matter as they are little more than unsightly lesions. The preservation of fair general health for many years, under proper care, is doubtless due to the striking lack of any tendency in this disease to involve viscera or blood-vessels, such as characterises ordinary rheumatism and gout - the former by endocarditis and pericarditis, and the latter in the way of arterio-sclerosis and cirrhoses of the renal and other organs. Anaemic individuals do poorly; and patients who present the greatest deformities of the lower extremities may have the least in the fingers, and are able to do much for themselves within the limitations of lying in bed continually.

C H I L D R E N are more amenable to treatment than adults and the aged, but in the latter the disease advances more slowly and with less permanent crippling. The affection is so rare, however, in the very young that it is difficult at times to say how the case will turn out. In severe cases the persistency of adolescence may be observed in the course of the disease, although it is capable of being modified by treatment and is attended with no immediate danger to life.

T R E A T M E N T.

GENERAL MEASURES.

The treatment of rheumatoid arthritis is usually rather disappointing; and perhaps no affection requires more perseverance and seld-reliance on the part of the physician or more hopeful resolution on that of the patient. The first thing to be done is to make an exhaustive search as to the probable cause of the disease, as its removal is an important step in the treatment of the affection, although such search is frequently futile, and many of the alleged causes may, after all, be mere antecedents or coincidences. However, inasmuch as the pathology of the affection is very obscure, any abnormal condition of organ or function thatmay be discovered should receive strict and prompt attention, lest it should, either through disturbed innervation or mal-

assimilation or impaired nutrition or defective excretion, be the predisposing or exciting cause of the disease. In women the most careful inquiry should be made into the condition of the ovario-uterine organs and functions, and the least departure from their norm should be treated forthwith. Deficient, excessive, or painful menstruation, leucorrhoea, ovarian irritations, or pain, even displacements of the uterus and ovaries, should be corrected as soon as possible. Repeated pregnancies and prolonged lactation, recurring mental anxiety and physical fatigue, defects of diet, want of food, of sunlight, and of good air, residence in damp swellings, occupations involving exposure to cold and wet, are conditions supplying important indications which too often are beyond the control of the physician, although they peremptorily require his attention. The general form is often met with in anaemic persons and those of impaired health and vigour, and probably very rarely occurs under opposite circumstances. In short, a patient presenting the symptoms of rheumatoid arthritis must be treated as an invalid with lowered vitality; and no greater mistake can be made than by treating him or her by depletion, as if the gouty condition existed. Aside from occasional simple local measures, the treatment should be almost wholly hygienic and dietetic. The patient should be removed from a damp locality or ill-ventilated apartment, and seek abundant fresh air, sunshine, good food, and cheerful surroundings, avoiding as far as possible all sources of anxiety and worry. He should dress warmly, wearing woollen next the skin and sleeping during the winter in flannels.

As regards diet, a reasonable variety may be allowed, so that it ample and nourishing, the chief dietetic indication being the avoidance of anything that will cause indigestion. From what has already been said, it will be obvious that a careful diagnosis should be made, so that the patient may receive a sustaining diet, and not the restricted feeding of the rheumatic and gouty. This is especially true as diet plays no part in the etiology of the disease. As in any chronic affection interfering with active exercise, the digestion is apt to be below the normal; and care must be taken that the food be digestible and taken in sufficiently small quantities. Heavy foods and indigestible articles must be avoided, as the patient must of necessity lead a sedentary life; as a result of the latter constipation is apt to exist, and calls for the usual laxative medicaments for its relief. Cereals, with fresh vegetables and fruits, and especially abundant animal food, should be eaten by the patient. Plenty of good milk, cream, butter, beef, mutton, poultry, eggs, and fish must make the basis of the menu. Alcohol has apparently no influence on the disease, and either spirituous or malt liquors, - ale, stout, porter, malt extracts, whisky, etc., - may be used, if desirable, with meals to support the patient's strength. In many cases, if the appetite fails from lack of ability to exercise in the open air, it is desirable to supplement the three regular meals of the day by ordering food between them, such as milk, egg-flip, and so forth.

MEDICINAL TREATMENT.

Internal medication is of little or no avail in any case of long standing, though numerous drugs have

been advised.

SALICYLATES.

It would seem that the salicylate of soda, given in sufficient doses, is not without utility, especially in the acute cases. Including the cases tabulated by Sée (Bull. de l'Acad. de Méd., Paris, 1877, v, S.2), Compagnon (On the Utility of Sadium Salicylate in the Treatment of Rheumatism, Paris, 1880) has described seventeen examples of rheumatoid arthritis, most of them of the general progressive form, in which great improvement as regards pain, stiffness, swelling, and even deformity, followed promplty the exhibition of this salt, even after the failure of other remedies. Various rebellious cases have also been benefited by others. Pollock has published an instance in which five grains of salicylate of quinine, three times a day were in three or four days followed by marked relief (Lancet, 1882, ii, 141). The testimony of other as to the great value of this salt in chronic rheumatism will be held by some to be corroborative of its value in rheumatoid arthritis. It is hardly necessary to say that it often fails in this intractable disease, but it has frequently relieved the pain and swelling and arrested the progress of it, at least for the time, even when failure has been experienced from the administration of the alklaies, iodine, and arsenic, and baths, etc. It is probable that less than forty-five grains daily of the sodium salt is of little value in even the most chronic forms, and that the quantity requires to be increased in proportion as the febrile symptoms are active, so that a drachm and a half or two drachms may need to be administered in the day to some persons. It should be given in divided doses at intervals of two hours, and, what is of primary importance, it should be continued for a long time, even after much improvement has resulted, and should be resorted to fromtime to time, especially during recurrences of the pain, heat, and swelling. Itis of consequence, especially in elderly patients, to ascertain that the drug is being promptly eliminated by the kidneys and to watch its effect upon the heart. The administration along with it of a little brandy or whisky of guaranteed purity will sometimes be necessary in feeble individuals. In those rather common cases in which the skin is inactive and perhaps harsh the salicylate often improves oxidation and elimination, and should it not do so the addition of the ammonium carbonate may be tried, especially in the debilitated and those whose cardiac action is enfeebled.

ASPIRIN.

This drug may be given in these cases, if preferred.

IODINE.

Various writers recommend iodide of potassium - in doses of ten grains three times a day well diluted in milk or Vichy. In combination with quinine or other tonic it will often prove signally useful in chronic cases unaccompanied by pyrexia, in which the pains are worst at night. It should at first be tried in moderate doses of five to eight grains, and be continued for a long time with occasional intermissions, and before discarding it from disappointment - which often arises - doses of fifteen to twenty grains may be given

tentatively for a short period.Milk or coffee or the mineral water above mentioned are good vehicles for its administration.

Whether free iodine in the form of the tincture,so highly spoken of by Lasègue(Arch.Gèn.de Mèd.,1856),acts as well or better than the iodide of potassium is doubtful.He gave it at meals,in doses progressively increased from ten minims to five or six grammes twice a day, in sherry or sweetened water,and persevered with it for a long period.

Some of the older writers reported restorations to health in severe forms of this disease from the persevering employment of the syrup of the iodide of iron.

IRON.

It is probable that the iron in the preparation just mentioned deserved as much commendation as the iodine,for it has often proved signally useful in this disease,not only on account of the anaemia which so frequently attends it,but through its beneficial influence upon the circulation and the general nutrition. The usual rules regulating the exhibition of iron are to be observed,and the condition of the digestive organs will demand special attention during its employment.

COD-LIVER OIL.

In chronic cases a prolonged course of cod-liver oil,alone or along with malt extract,often seems to be of real service,especially when nutrition is much impaired,or when the patient is the subject of inherited or acquired tuberculous taint.It should be given in small doses at first,and gradually increased to the full dose as the case may require.

QUININE.

Quinine is sometimes of service in the more acute cases,and may be usefully combined with belladonna and strychnine for the control of the perspirations.

ARSENIC.

Although the influence of arsenic upon rheumatoid arthritis is not uniform,yet it has sometimes proved useful,and may be tried in suitable cases.Bardsley(Medical Reports,1807),Begbie(Edin.Med.& Surg.Jour., 1858),Fuller(loc.cit.),Garrod(loc.cit.),Gueneau de Mussy(Bull.de Thèrap.,1864,lxvii,p.24),Charcot(loc.cit.), and numerous others speak favourably of its employment. Like iron,it may prove beneficial in various ways: by improving the quality of the blood,promoting the circulation in the superficial layers of the skin,or exerting some influence upon either the nerve-centres or perhaps upon the vaso-motor nerves of the cutaneous or articular tissues.The last-mentioned suggestion is favoured by the circumstance noted by Charcot,namely,that the first effects of arsenic in arthritis deformans are often intensification of the articular pains,and somstimes the production of redness and swelling where they did not exist before.Some writers have found arsenic without effect or injurious in very inveterate cases,and when the disease had appeared at an advanced age. Five to ten minims of Fowler's solution,or of the solution of the arseniate of sodium,which is perhaps less irritating than the former,should be given immediately after meals,and its effects upon gastric and hepatic functions carefully watched.It may be given with iron or with the syrup of the iodide of that metal.De Mussy

has highly recommended arsenical baths consisting of a half to two drachms of arseniate of soda to thirty gallons of water; but any improvement which may follow its employment is probably owing to the temperature of the bath or the bath itself, as arsenic is not absorbed by the unbroken skin.

ANTIPYRETICS.

Phenacetine and any of the other antipyretics may be administered for the relief of the pain and fever in acute cases, with due regard, of course, to the condition of the heart and other indications arising.

OPIUM.

Cases temporarily relieved b y other medicaments may require recourse to opium; but in chronic cases the danger of establishing a taste for the drug should be borne carefully in mind.

ALKALIES.

Charcot (loc.cit.) recommended the administration of carbonate of sodium to the amount of about an ounce, in divided doses, each day for a number of weeks. According to my experience, the alkalies are worse than useless - the so-called cure being nearly worse than the disease.

MISCELLANEOUS MEDICAMENTS.

Those who refer rheumatoid arthritis to a bacterial origination tell us that the therapeutic measures should be carefully aimed at the infection by microbes and the toxins which they elaborate, remedies capable of strengthening the forces and vital energies of the patient being necessary as well. As powerful antidotes against bacterial invasion, the creosotes, naphthols, and phenols, are recommended as valuable. Bannatyne prefers creosotal, and most of all the guaiacol-carbonate. Creosotal is a think fluid, soluble in alcohol, but not in water; it has an oily taste, and a rather nasty odour, and is given in doses of five or eight minims three times a day. Guaiacol carbonate is a white crystalline, insoluble, tasteless and odourless powder, which in the intestines is decomposed, yielding guaiacol; it is given in doses of five to eight grains three to six times a day. The exhibition of the drug should be continued for a long time, and its advocates claim for it excellent results; it alleviates, they say, the pain, and has an effect upon the morbid process itself. Benzosol (benzoyl-guaiacol) may be employed if preferred. It appears also that Zolotavin (La Méd.Mod., Sept.18, 1897) has used lactic acid with success in an old case of rheumatoid arthritis. Ten drops of this drug were administered upon an empty stomach, no food being allowed for an hour afterwards or more. His results lack confirmation, however. Guaiacum has been recommended, but is of little use, except as a means of stimulating the circulation and promoting moderate diaphoresis. Colchicum is useless.

BATHS.

The value of baths in the treatment of rheumatoid arthritis seems well established, both natural and artificial ones being employed. Their importance was long ago pointed out especially by Niemeyer (Text-Book Prac.Med., 1867, p.488) and Bouther (On the Treatment of Rheumatism by Baths of High Temperature, Paris, 1878). It is neither the nature or the proportion of their mineral ingredients, but the degree of temperature,

which constitutes the essential point in the action of the bath.This,if true,explains the almost equal reputation of many varieties of thermal springs in the treatment of rheumatoid arthritis and chronic rheumatism. It is this that permits the physician to promise the sufferer as much benefit from the employment of baths of simple water as those of the most noted health resorts abroad.The time for a resort to hot baths in rheumatoid arthritis is when the very violent pains have subsided sufficiently to allow of their employment;and while they may be hopefully used in the most chronic and advanced cases,the earlier they are employed the more curative they are.The temperature of these hot baths need not,as a rule,exceed 95.to 100.F.,although some authorities approve of raising the temperature to 110.or 112.F.while the patient is in the water.A series of twenty to thirty such baths,taken every second day for ten to twenty minutes,is sufficient for one trial,and very often effects marked inprovement in the disease.The aggravation or return of pain in the joints which often follows the employment of warm baths will cease after the fifth or sixth bath.The Turkish bath is not to be recommended; it very often does much mischief by causing debility,and some of the older writers affirm that its excessive use has induced arthritis deformans in persons previously free from the disease.Now,while it may be true that simple hot-water baths employed at home are as good as mineral thermal baths taken at their source,it is generally admitted that it is best to send persons who can afford the expense to the springs themselves,where they may drink the waters as well as employ them externally,and at the same time secure all advantages arising from change of habits,scene,and climate,from restriction to a proper diet,and from the systematic employment of the waters and baths under the direction of persons experienced in their administration,etc.No reliable rules can be alid down for the selection of the mineral waters best adapted to each case;the stronger alkaline waters perhaps had better be used with great care, such as those of Carlsbad,Vichy,Mount Doré,Weisbaden, Bath,Aix-la-Chapelle,etc.;some writers recommend resort to some place where the air is bracing and the waters tonic or chalybeate,as Buxton,Spa,Schwalbach,or St. Moritz.In the United States of America good results are obtained at the Hot Springs of Arkansas and the Hot Sulphur and the Lithia Springs of Virginia.The use internally and in the form of hot baths of the mineral springs abroad is frequently beneficial.In the selettion of the mineral waters to be drunk,and of the temperature and other qualities of the mineral-waters baths employed,careful attantion must be paid to the condition of the functions of the skin,liver,kidneys,and nervous system.Moreover,it occasionally happens that after failure of sulphur or alkaline baths some other form may succeed,as the vapour or hot-air,or tepid or very hot water-bath.If decided benefit follow the first series of baths,recourse should be had from time to time to a fresh series,even for several years,in obstinate cases. Mud and peat baths are much valued on the Continent, but aged or feeble individuals do not bear them well. The course of treatment at the various institutions so largely advertised should be taken at least once a year,but only in the early stages of the disease,or

more harm than good may be done. Wood (Med. News, July 17, 1897) says that the local hot, dry-air bath is of little value in rheumatoid arthritis, but that it is of great service in the treatment of the ligamentous inflammations and in the tenosynovitis of the disease; the temperature he considered of most value was the one ranging between 270. and 320. F.

LOCAL TREATMENT.

This does not yield in importance to any other, and is not infrequently more effective in restoring the functions of the articulations than the local. In that rare variety, acute rheumatoid arthritis, attended with much pain and heat in the joints, perfect rest in bed is called for, together with the other measures adapted to subdue the inflammation and allay the pain.

Compresses, wet with warm water, rendered anodyne by the addition of laudanum or belladonna, or both, and covered with oilsilk, suit some cases - light linseed poultices, applied moderately warm and extending considerably beyond the limits of the articulations and covered with guttapercha or oilsilk, in others.

As the pain and local heat, the tincture of iodine may be freely applied, or blisters, solid or liquid, over limited areas above and below the affected joints, but not on them until the inflammation has very much abated and is becoming chronic. These simple methods should be employed assiduously and be aided by appliances to secure perfect rest to the inflamed articulations.

In the chronic variety complete rest is not needed unless during the acute exacerbations, but the movements should be at first somewhat restrained and be regulated by the effects produced. But the severe pain experienced during the movements must be borne; it will subside promptly. Decided increase of pain and heat in the part, lasting many hours, would indicate more reserve in the use of the joints. It is frequently very difficult to determine when and to what extent movement may be permitted in this disease. No fixed rule can be laid down of universal application, but it may be stated that in proportion as the local disease becomes indolent and inactive may pressure and active movements of the joints be resorted to; for they then have a beneficial influence in preventing stiffness, contraction, and deformity. Indeed, it would seem inadvisable to delay these movements long even in subacute cases. Some writers have especially insisted upon the importance of systematic daily movements of the affected joints as the most essential point of the treatment, combined with thorough massage of all the muscles whose functional activity is impeded and impaired.

The stiffness of the chronic cases may be relieved more by hydrotherapy with judiciously applied massage or Swedish movements than in any other way; but all such treatment must be mild at first or it will make matters worse; and the patient must understand that it must be continued for fully a month or six weeks before very decided results are obtainable. Gentle friction with oil or lanolin tends to promote the absorption of effusions and increase the motile power of the part.

There are various ways of treating the abiding chronic inflammation indicated by local heat, swelling, and inflammation of the affected tissues. The joints may be thoroughly fomented with tolerably hot water

or by means of the local vapour bath for half an hour, night and morning, and then be gently rubbed for ten or fifteen minutes with iodine or weak mercurial ointment or with the compound camphor or acetic turpentine liniment, or, of these are too stimulating, with some bland oil, such as cod-liver or neats'-foot or cocoa-oil, after which should be applied hot-water compresses or linseed poultices or a wrap of soft cottonwool covered with oil-silk and secured by an elastic, moderately-tight roller. It is a common experience to find benefit follow the daily soaking of the affected articulations for ten minutes in water as hot as can be borne, accompanied by massage; very considerable improvement may be observed in the reduction of ligamentous swelling and fluid accumulation, though obviously exostoses cannot be influenced by such treatment. Patients who are fairly strong derive improvement from the Scotch douche, that is, alternate hot and cold water douching under considerable pressure. The local hot-air bath is of signal service in not a few cases. The joints are enclosed in an adjustable box, made to fit almost any kind of joint, and air heated by a lamp to 180. or 200. F. is passed around them for about forty-five minutes. If a cloth be laid over the joints, it becomes moistened with the intense perspiration excited and acts veru much like a poultice, so that for an hour or two after removal of the apparatus the skin llooks almost parched; it soon regains its normal appearance, however. Haygarth, Strümpell, and other extol the use of hot sand. General hot-water or vapour baths are too debilitating for advanced cases, and their use should be condemned.

Should these measures prove ineffective and the inflammatory process grow more indolent, counter-irritants may be conjoined with or substituted for them. Small fly blisters or strong iodine paint may be applied close to the joints, or the ordinary tincture of iodine may be brushed over them, or the above ointments or liniments and one of the bland oils may be more forcibly rubbed in. Prolonged massage merits more appreciation than it has hitherto received. Methyl salicylate has also been tried. Bannatyne advocates the external use of guaiacol, in combination with an equal amount of olive-oil or combined with tincture of iodine - six parts of guaiacol to one part of iodine; the odour of the guaiacol can be masked by a few drops of the oil of cloves, the mixture being painted on the affected part and covered with a dry dressing.

The above list does not exhaust the various medicaments that have from time to time been advised. Robinson (Merck's Arch., April, 1902) uses a paint composed of menthol (one drachm), salicylic acid (two drachms), methyl salicylate (one drachm), and alcohol (sufficient to make an ounce). This he applies with a camel's-hair brush briskly to the affected parts, covering same with absorbent cottonwool and oilsilk, and lightly bandaging. He tells us that so efficient has this paint of his become that it is in the vast majority instances possible to dispense with internal remedies altogether. It is applicable in acute cases mainly, and the salicylic acid and methyl salicylate are rapidly absorbed and their presence can be demonstrated in the urine. In this way he is enabled to saturate the system with salicylates without disturbing the gastric function. After a few applications the epidermis begins to peel off, and the surface

becomes tender.When this occurs the application should be stopped for a day or two,and an emollient ointment should take its place.In subacute cases the efficiency of the remedy is very slight,while in chronic cases it is generally useless. In these circumstances he says he has obtained the best results from a thirty-three per cent.ichthyol ointment or a twenty per cent.ichthyol-glycerine solution,and long-continued internal administration of ichthyol and potassium iodide.

The use of electricity has been advised,but not a few observers affirm that it is not so useful in rheumatoid arthritis as in gout.I have used it largely,and find that in the chronic as well as in the earlier stages it is an important adjuvant,not only improving the nutrition of the muscles,but in promoting absorption, allaying pain,and subduing excitability of peripheral structures,removing muscular contractions,and probably modifying the local inflammatory processes.It gives me the impression in some cases of improving the general health.The constant current is generally the most useful, and should have an intensity of from about ten to fifteen milliampères,and be applied daily for ten or fifteen minutes.The positive pole,terminating in a large flat moistened sponge,is applied to the spinal origin of the brachial or lumbar plexus,according as the superior or inferior extremities suffer,while the negative pole is immersed in a vessel of warm salt water in which the hands or feet are placed.Some apply the negative electrode to the joints and the positive to the limb higher up.The faradic current may also be employed on account of its action upon the muscles and small vessels.In the advanced stages attended with marked thickening of the articular and periarticular tissues,with contraction of the muscles and greater or less impairment of movement, the above measures sre still our chief resources;but they mat be employed more vigorously.

There is in these very chronic and incurable cases little or no risk of lighting up inflammation;we indeed desire to excite a more active circulation in the part with a view of removing the congested state of the capillaries and venules,so favourable to the development of fibrous growths.In this stage especially vigorous and active and passive movements of the affected joints,and massage of the muscles which move them,and gymnastics, are imperatively needed;and it is sometimes almost marvellous what an amount of mobility and usefulness may thereby be restored to apparently crippled and deformed articulations and extremities.Persons who have not walked for years are frequently so much improved as to be able to leave their sofa or bed,and with or without crutches or mechanical aids walk about,while their abiding pains depart,and this notwithstanding the permanent deformity of the articular surfaces.

Finally,we may note that continental writers have advised a surgical treatment treatment of the disease. Thus,Schüller claims to have obtained a complete cure and restoration of absolutely normal movements by treating the affected articulations with repeated injections of a mixture composed of twenty parts of iodoform,two hundred and fifty to four hundred parts of acid-free glycerine,and five parts of guaiacol - the operations being performed,of course,under strict antiseptic precautions.Usually the pain continues for

some days,but fever only exceptionally results.Twenty-seven cases were treated in this manner,and of these sixteen recovered almost completely. But Schüller adds that the cure is more rapidly effected by incision of the affected joint,removal of the diseased synovial membrane and the villous mass,and suturing of the wound with injection of the above-described mixture into the articulation.The joint is kept immovable for ten days and extended by a bandage.After that the sutures and the bandage are removed,and recourse is had to massage, electricity,inunctions,and baths.

In the case of C H I L D R E N the treatment is conducted on similar principles, bearing particularly in mind the fact that all lowering or drastic management is both useless and injurious. Thus,serious aggravation of all the symptoms may follow severe treatment by purgatives,alkalies,and colchicum,with low diet;while substitution for this of tonics and a liberal dietary may produce a remarkable improvement.Everything that tends to improve general health and nutrition is beneficial in this disease.A warm,sunny climate,simple nutritious food,and warm clothing are of the first importance.Locally,as well as generally,all severe measures are harmful.In children the joints will not bear blisters or strong stimulating applications or counter-irritants,which tend to increase the morbid changes in and around the joints.A weak of solution of iodine with glycerine,fifteen minims of the former to sixty of the latter in an ounce of water,maybe applied under oiled silk or wool,but nothing more,though in certain cases Bannatyne's guaiacol and olive-oil in equal parts may be tried.Gentle rubbing and exercise of the joint help to keep the movement free.Children with rheumatoid arthritis sometimes respndd satisfactorily to the exhibition of iodide of potassium and arsenic,and the most of them do well on cod-liver oil and other tonics.The progress of the disease is not infrequently arrested by a w/inter residence in a warm climate,such as that of Cairo or Tangiers.A mere change of scene and climate is often beneficial by improving the general health and occupying the mind with new interests,and keeping the patient in the best of spirits. Of all the forms of medication that have been recommended the most successful may be considered that by the daily use of warm and other baths,with the administration of the syrup of the iodide of iron in increasing doses until the limit of comfortable toleration by the stomach has been reached.

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